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Dr. Richard A. Reeve.....22 Shuter St.....Toronto, Canada.....
Dr. Henry O. Reik.....506 Cathedral St.....Baltimore, Md.....
Dr. W. Scott Renner.....341 Linwood Ave.....Buffalo, N. Y.....
Dr. George L. Richards.....84 North Main St.....Fall River Mass.....
Dr. Huntington Richards.. St. Paul's School.....Concord, N. H.....
Dr. John D. Richards*.....44 West 49th St.....New York, N. Y.....
Dr. Charles W. Richardson..1317 Connecticut Ave.....Washington, D. C.....
Dr. Stephen O. Richey.....732 17th St.....Washington, D. C.....
Dr. Charles E. Rider.
Dr. Wheelock Rider.....53 South Fitzhugh St.....Rochester, N. Y.....
Dr. F. W. Ring.
Dr. Henry W. Ring.....187 Church St.....New Haven, Conn....
Dr. Samuel D. Risley.....1738 Chestnut St.....Philadelphia, Pa.....
Dr. Edmund C. Rivers.....1632 Welton St.....Denver, Col.....
Dr. Arthur W. de Roaldes...624 Gravier St.....New Orleans, La.....
Dr. Walter Roberts*.....1732 Spruce St.....Philadelphia, Pa.....
Dr. William K. Rogers*....88 East State St.....Columbus, Ohio.....
Dr. D. B. St. John Roosa.
Dr. John D. Rushmore.....129 Montague St.....Brooklyn, N. Y.....
Dr. Wm. W. Seely.
Dr. Ralph W. Seiss.....255 South 17th St.....Philadelphia, Pa.....
Dr. S. Sexton.
Dr. George E. Shambaugh..100 State St.....Chicago, Ill.....
Dr. John B. Shapleigh.....Grand & Washington Aves..St. Louis, Mo.....
Dr. Warren S. Shattuck, Jr..160 Clinton St.....Brooklyn, N. Y.....
Dr. Henry L. Shaw.....19 Commonwealth Ave.....Boston, Mass.....
Dr. John E. Sheppard.....130 Montague St.....Brooklyn, N. Y.....
Dr. E. Terry Smith.....75 Pratt St.....Hartford, Conn.....
Dr. S. MacCuen Smith*.....1429 Spruce St.....Philadelphia, Pa.....
Dr. Sargent F. Snow*.....707 University Block.....Syracuse, N. Y.....
Dr. Edmund D. Spear.....100 Boylston St.....Boston, Mass.....
Dr. Horatio N. Spencer.....2725 Washington Ave.....St. Louis, Mo.....
Dr. Selden Spencer*.....2725 Washington Ave.....St. Louis, Mo.....
Dr. Frank Sprague.
Dr. Franklin M. Stephens*..71 West 49th St.....New York, N. Y.....
Dr. George T. Stevens.....22 East 46th St.....New York, N. Y.....
Dr. Samuel B. St. John.
Dr. Edward Stieren*.....3603 Fifth Ave.....Pittsburg, Pa.....
Dr. George Strawbridge....202 South 15th St.....Philadelphia, Pa.....
Dr. Joseph A. Stucky.....Lexington, Ky.....

Dr. Theoron Y. Sutphen....999 Broad St.....Newark, N. J.....
Dr. Lewis H. Taylor.....82 South Franklin St.....Wilkes Barre, Pa.....
Dr. Samuel Theobald.....Cathedral & Howard Sts....Baltimore, Md.....
Dr. William Thomson.
Dr. Robert Tilley.
Dr. Andrew Timberman*...112 East Broad St.....Columbus, Ohio.....
Dr. John Van Duyn.....318 James St.....Syracuse, N. Y.....
Dr. J. J. B. Vermyne.
Dr. Ernest De Wolf Wales..423 Board of Trade Bldg.. Indianapolis, Ind.....
Dr. D. Harold Walker.....28 Newbury St.....Boston, Mass.....
Dr. David Webster.....308 Madison Ave.....New York, N. Y.....
Dr. Joseph A. White*.....200 East Franklin St.....Richmond, Va.....
Dr. Fred Whiting.....19 West 47th St.....New York, N. Y.....
Dr. Frederick M. Wilson...834 Myrtle Ave.....Bridgeport, Conn.....
Dr. Hiram Woods.....842 Park Ave.....Baltimore, Md.....

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*Associate.

American Otological Society.

The Forty-third Annual Meeting of the American Otological Society was held at the Hotel Arlington, Washington, D. C., May 3 and 4, 1910.

The following members were present:

DRS. ALPHEUS E. ADAMS.....Newburg, N. Y.
JOHN L. ADAMS.....New York, N. Y.
T. PASSMORE BERENS.....New York, N. Y.
HERBERT S. BIRKETT.....Montreal, Canada.
WILLIAM C. BRAISLIN.....Brooklyn, N. Y.
JOSEPH H. BRYAN.....Washington, D. C.
W. SOHIER BRYANT.....New York, N. Y.
RALPH BUTLER.....Philadelphia, Pa.
CORNELIUS G. COAKLEY.....New York, N. Y.
J. FRANK CROUCH.....Baltimore, Md.
JOHN P. DAVIDSON.....Richmond, Va.
EWING W. DAY.....Pittsburg, Pa.
EDWARD B. DENCH.....New York, N. Y.
ARTHUR B. DUEL.....New York, N. Y.
JOHN DUNN.....Richmond, Va.
GEORGE F. FISKE.....Chicago, Ill.
PERCY FRIDENBERG.....New York, N. Y.
HARRY FRIEDENWALD.....Baltimore, Md.
EMIL GRUENING.....New York, N. Y.
THOMAS H. HALSTED.....Syracuse, N. Y.
HERBERT HARLAN.....Baltimore, Md.
DAVID HARROWER, JR.....Worcester, Mass.
WILLIAM H. HASKIN.....New York, N. Y.
ERASTUS E. HOLT.....Portland, Me.
FREDERICK L. JACK.....Boston, Mass.
WALTER B. JOHNSON.....Paterson, N. J.
RICHARD JORDAN.....New York, N. Y.
JOSEPH A. KENEFICK.....New York, N. Y.

BEVERLEY R. KENNON.....Norfolk, Va.
CHARLES J. KIPP.....Newark, N. J.
D. BRADEN KYLE.....Philadelphia, Pa.
GEORGE A. LELAND.....Boston, Mass.
WILLIAM LINCOLN.....Cleveland, Ohio.
FRANCIS B. LORING.....Washington, D. C.
DAVID B. LOVELL.....Worcester, Mass.
CHARLES H. MAY.....New York, N. Y.
ROBERT C. MYLES.....New York, N. Y.
JAMES F. McKERNON.....New York, N. Y.
FRANCIS R. PACKARD.....Philadelphia, Pa.
WENDELL C. PHILLIPS.....New York, N. Y.
NORVAL H. PIERCE.....Chicago, Ill.
JOHN B. RAE.....New York, N. Y.
B. ALEXANDER RANDALL.....Philadelphia, Pa.
J. M. RAY.....Louisville, Ky.
RICHARD A. REEVE.....Toronto, Canada.
HENRY O. REIK.....Baltimore, Md.
W. SCOTT RENNER.....Buffalo, N. Y.
GEORGE L. RICHARDS.....Fall River, Mass.
CHARLES W. RICHARDSON.....Washington, D. C.
STEPHEN O. RICHEY.....Washington, D. C.
WHEELOCK RIDER.....Rochester, N. Y.
HENRY W. RING.....New Haven, Conn.
JOHN E. SHEPPARD.....Brooklyn, N. Y.
GEORGE E. SHAMBAUGH.....Chicago, Ill.
E. TERRY SMITH.....Hartford, Conn.
S. MacCUEN SMITH.....Philadelphia, Pa.
GEORGE T. STEVENS.....New York, N. Y.
EDWARD STIEREN.....Pittsburg, Pa.
THEORON Y. SUTPHEN.....Newark, N. J.
LEWIS H. TAYLOR.....Wilkes-Barre, Pa.
D. HAROLD WALKER.....Boston, Mass.
JOSEPH A. WHITE.....Richmond, Va.
FRED WHITING.....New York, N. Y.
FREDERICK M. WILSON.....Bridgeport, Conn.
HIRAM WOODS.....Baltimore, Md.

The meeting was called to order at 10 a. m. by the president, Dr. Frederick L. Jack, of Boston, Mass., who called attention to a change in the By-Laws to the effect that unless a member attends one meeting in three years he is dropped from the roll of the Society. This does not include members of twenty years' standing.

The chair then appointed the following members to act as the Business Committee: Drs. Gruening, New York; Richards, Fall River; Whiting, New York.

It was moved, seconded and carried that the Treasurer's report be accepted and placed on file.

The Committee on Membership reported favorably on the following names: Drs. James F. McCaw, Watertown, N. Y.; Joseph A. Stucky, Lexington, Ky.; Philip D. Kerrison, New York, N .Y.; Thomas Hubbard, Toledo, O.

It was recommended that the names of Drs. Page and McCoy be carried over for further consideration at the next meeting.

The following candidates were then nominated for membership: Dr. Max A. Goldstein, St. Louis, proposed by Dr. G. L. Richards, seconded by Dr. Walter B. Johnson; Dr. B. L. Shurly, Detroit, proposed by Dr. J. E. Logan, seconded by Dr. D. Braden Kyle; Dr. H. P. Mosher, Boston, proposed by Dr. G. L. Richards, seconded by Dr. F. L. Jack; Dr. Harris G. Sherman, Cleveland, proposed by Dr. William Lincoln, seconded by Dr. W. S. Bryant; Dr. Seymour Oppenheimer, New York, proposed by Dr. E. Gruening, seconded by Dr. Arthur B. Duel; Dr. Hanau W. Loeb, St. Louis, proposed by Dr. Arthur B. Duel, seconded by Dr. E. Gruening.

Honorary Fellows: Dr. Herman Knapp, New York, proposed by Dr. E. B. Dench, seconded by Dr. B. A. Randall; Dr. J. S. Prout, Brooklyn, proposed by Dr. B. A. Randall, seconded by Dr. H. O. Reik.

The reception of guests then took place, the following being present: Dr. J. Kerr Love, Glasgow, Scotland; Dr. Seymour Oppenheimer, New York; Dr. James G. Gwyer, New York; Dr. John A. Welch, New York; Dr. Willard G. Reynolds, Brooklyn; Dr. Stephen H. Lutz, Brooklyn; Dr. J. A. Stucky, Lexington, Ky.; Dr. O. A. H. McKimmie, Washington, D. C.; Dr. Grant Selfridge, San Francisco, Cal.; Dr. Perry Goldsmith, Toronto, Canada; Dr. C. A. Clapp, Baltimore, Md.

It was moved, seconded and carried that guests be invited to take part in the discussions.

Dr. Emil Gruening, New York, read a paper on Pediatrics and Otiatrics.

Dr. Fred Whiting, New York, read a paper on Rapid Fluctuations of Temperature in Otitis Media of Infants and Young Children as an Indication for Mastoid Operation.

Discussed by Dr. Charles Gilmore Kerley, New York; Dr. Linnaeus E. La Fetra, New York; Dr. Edward B. Dench, New York; Dr. George L. Richards, Fall River; Dr. Arthur B. Duel, New York; Dr. James F. McKernon, New York; Dr. B. Alexander Randall, Philadelphia; Dr. E. Gruening, New York; and in closing by the essayist.

Dr. Cornelius G. Coakley, New York, read a paper on Observations upon the Association of Acute Suppurative Otitis Media and Acute Suppurative Inflammation of the Nasal Accessory Sinuses in Adults.

Discussed by Dr. William H. Haskin, New York; Dr. George L. Richards, Fall River; Dr. John E. Sheppard, Brooklyn; Dr. Fred Whiting, New York; and in closing by the essayist.

Dr. John E. Welch, New York, read a paper on Unsuspected Middle Ear Conditions Found in Routine Post-Mortem Examinations.

Discussed by Dr. Joseph A. Kenefick, New York; Dr. Wendell C. Phillips, New York; Dr. George F. Fiske, Chicago; and in closing by the essayist.

Dr. John E. Sheppard, Brooklyn, read a paper on (a) Some Cases of Otitic Meningitis; (b) A Case of Tympanic Vertigo.

Discussed by Dr. Edward B. Dench, New York; Dr. Norval H. Pierce, Chicago; Dr. Wendell C. Phillips, New York; Dr. James F. McKernon, New York; Dr. S. MacCuen Smith, Philadelphia; Dr. Frederick L. Jack, Boston; and in closing by the essayist.

The following motion by Dr. E. B. Dench was seconded and carried: That the Chair be authorized to appoint a committee of three whose duty it shall be to endeavor to secure in every medical school in the United States and Canada, the delivery of at least one lecture during the

course on the educational problem presented by the deaf child.

The Chair appointed the following members to serve as such a Committee: Dr. E. B. Dench, New York; Dr. G. Hudson Makuen, Philadelphia; Dr. Herbert S. Birkett, Montreal, Canada.

Morning Session, May 4, 1910.

The meeting was called to order at 10 a. m., Dr. Frederick L. Jack, presiding.

Letters of resignation were presented from Dr. Herman Knapp, New York; and Dr. J. A. Lippincott, Pittsburg.

The death of the following members was announced: Dr. Gordon King, New Orleans; Dr. Samuel B. St. John, Hartford; Dr. Frank B. Sprague, Providence.

Dr. Edward B. Dench moved that the Chair appoint a committee of three to draw up appropriate resolutions concerning the death of these members and that copies of the same be forwarded to their respective families. The motion was seconded and carried.

The following committee was appointed. Dr. E. B. Dench, Dr. J. F. McKernon, and Dr. B. A. Randall.

The Standing Committee on Program and Place of Meeting was then appointed, and consisted of the in-coming President, Secretary, and Dr. H. O. Reik.

The Committee on Inviting The International Otological Society to meet in America submitted the following report:

"Your Committee appointed in 1908 to invite the International Congress of Otology to meet in America, drafted and had suitably engrossed such an invitation embodying the later instructions voted in 1909, that New York be named as the place of meeting. Formal invitations were likewise presented from Boston and Philadelphia and from the American Otological, Laryngological and Rhinological Societies and considered by the Committee of Organization, in whose hands the decision lay. On motion of Urban

Pritchard, it was voted that the Congress come to America in 1912. By a second vote as to the place of meeting, Boston was almost unanimously chosen. The details as to the exact date, etc., were left to the American Organization Committee of one, Dr. Clarence J. Blake."

B. Alexander Randall,
Vice-Chairman.

Dr. William H. Haskin, New York, read a paper on The Vacuum Cleaner. Its Applicability in Routine Office Practice and in Operative Work for Sponging. Description of Apparatus.

Dr. George E. Shambaugh, Chicago, read a paper on (a) Distribution of the Blood Vessels in the Labyrinth of the Human Ear; (b) Demonstration of Preparations Showing the Blood Vessels in the Human Ear and in the Labyrinth of the Dog's Ear.

Dr. Norval H. Pierce, Chicago, read a paper on Two Cases of Labyrinthine Involvement in Suppurative Ear Diseases Occurring During the Year.

Discussed by Dr. Edward B. Dench, New York; Dr. Arthur B. Duel, New York; Dr. George E. Shambaugh, Chicago; and in closing by the essayist.

Dr. William C. Braislin, Brooklyn, read a paper on The Maxillary and Ethmoidal Spaces in the Five Year Old Child.

Dr. Wendell C. Phillips, New York, read a paper on Report of a Case of Tuberculous Meningitis Following Chronic Otitis Media and Complicated by Measles and Anterior Poliomyelitis.

Dr. E. Terry Smith, Hartford, read a paper on The Present Status of the Adenoid Operation in Children.

Discussed by Dr. Hiram Woods, Baltimore; Dr. Robert C. Myles, New York; Dr. Wendell C. Phillips, New York; Dr. James Kerr Love, Glasgow; Dr. George A. Leland, Boston; Dr. B. Alexander Randall, Philadelphia; Dr. Frederick L. Jack, Boston; Dr. William C. Braislin, Brooklyn; Dr. George E. Shambaugh, Chicago; and in closing by the essayist.

Dr. Charles H. Richardson, Washington, read a paper on Report of a Case of Osteomyelitis of the Temporal Bone.

Discussed by Dr. Hiram Woods, Baltimore; Dr. Edward B. Dench, New York; Dr. B. Alexander Randall, Philadelphia; and in closing by the essayist.

Dr. Hiram Woods, Baltimore, read a paper on Review of Some Cases of Acute Otitis Media with Uncommon Symptoms.

Discussed by Dr. E. Terry Smith, Hartford; Dr. Edward B. Dench, New York; Dr. Ewing W. Day, Pittsburg; and in closing by the essayist.

Dr. W. Sohier Bryant, New York, read a paper on A Case of Mastoiditis, Complicated by Septic Pericarditis, Resulting Fatally.

Discussed by Dr. E. B. Dench, New York.

Dr. D. Harold Walker, Boston, read a paper on Ménière's Disease. Report of a Series of Cases Occurring in One Family.

Discussed by Dr. George E. Shambaugh, Chicago; Dr. Hiram Woods, Baltimore; and in closing by the essayist.

Dr. George L. Richards, Fall River, read a paper on Erysipelas as a Complication of Mastoid Disease.

Discussed by Dr. Wendell C. Phillips, New York; Dr. C. G. Coakley, New York; Dr. B. Alexander Randall, Philadelphia; Dr. Walter B. Johnson, Paterson; Dr. George A. Leland, Boston; and in closing by the essayist.

Dr. Jesse Wright Downey, Jr., Baltimore, read a paper on A Further Clinical Study of the Vascular Theory of Tinnitus Aurium, by title.

Dr. S. MacCuen Smith, Philadelphia, read a paper on Report of Two Cases of Cerebellar Abscess, by title.

Dr. D. Braden Kyle, Philadelphia, read a paper on Psychological Deafness, by title.

Afternoon Session, May 4, 1910.

The meeting was called to order at 1 p. m., Dr. Frederick L. Jack presiding.

The Nominating Committee reported as follows:

President—Dr. Edward Bradford Dench.

Vice-President—Dr. Charles W. Richardson.

Secretary and Treasurer—Dr. James F. McKernon.

Membership Committee—Dr. Frederick L. Jack, Dr. Norval H. Pierce, Dr. Henry O. Reik.

Publication Committee—Dr. Wendell C. Phillips, Dr. B. Alexander Randall, the Secretary, ex-officio.

It was moved, seconded and carried that the Secretary cast one vote for the officers as nominated. The Secretary so voted.

There was some discussion about the expense of printing charts, etc., but this did not take the form of a motion.

Dr. B. Alexander Randall, Philadelphia, and Dr. Henry O. Reik, Baltimore, presented a paper on Vaccine Therapy.

JAMES F. McKERNON,
Secretary.

PEDIATRICS AND OTIATRICS.

By EMIL GRUENING, M. D., New York, N. Y.

Pediatrics is not a specialty. It is the field of general medicine applied to the infant and child. It summons all specialties to its aid, and therefore also has need of otiatrics.

The otology of today is a science resting securely on anatomical, physiological, and pathological bases, and using rational therapeutic methods to meet the clinical conditons. This is a development of the last fifty years, and in this connection we have to mention gratefully the names of Toynbee, v. Troeltsch, Schwartze, Politzer, Zaufal, Macewen, and others. Let us now consider what bearing this new science has on the current practice of pediatrics.

The literature which I studied with this in view gives otology full credit for its present standing. In the "Traité des Maladies de l'Enfance" by Grancher and Comby, the chapter, "Les Maladies des Oreilles," has been written by an otologist of note, Moure of Bourdeaux. In the five volumes edited by Hutinel, "Les Maladies des Enfants," there is no special chapter on the otology of children, and ear disease is there treated only as it occurs in connection with other diseases. The same remark applies to the "Handbuch der Kinderheilkunde," by Pfaundler and Schlossman.

Of the American literature I reviewed first the "Collectanea" of Abraham Jacobi, who is abreast of modern thought in his otology as in all other subjects. I have also looked through the last ten volumes of the "Archives of Pediatrics," at present issued by La Fetra. We find during the ten years some abstracts of otological papers, a few minor articles by otologists, and but two important studies by pediatrists, one by Dr. Huber on "Otitic Serous Meningitis and Its Treatment by Lumbar Puncture," the other by

Dr. Kerley, on "Acute Otitis in Children," fifty-one cases observed in private practice. In forty-four of these the otitis was uncomplicated, four presented a mastoid involvement requiring operation, and in three there was an implication of the jugular bulb. From the scantiness of articles on the subject written by pediatrists in current literature, it would seem that aural examination is not as widely practised and written on as the subject demands. Of American text-books on the diseases of children I looked over: "Diseases of Infancy and Childhood" by Emmet Holt, "Diseases of Infancy and Childhood" by Henry Koplik, "Treatment of the Diseases of Children" by Charles Gilmore Kerley, "Diseases of Infancy and Childhood" by Louis Fischer, and "Diseases of Infants and Children" by Chapin and Pisek.

All these authors accept the teaching of modern otology, though Holt makes decided reservations as to the indications for operative procedure. Holt considers the mastoid operation as one fraught with danger, especially for infants, and advises delay until the indications are imperative. He thinks that the operation is performed too frequently, and often with insufficient indications, and that serious intracranial complications but rarely occur.

It is unnecessary in this paper to state what are the indications for the mastoid operation. The criticism by Dr. Holt of otiatric surgery and its votaries does not agree with my experience. I have operated on mastoid in public in several large hospitals, general and special, in the presence of practitioners and students, for more than thirty years. I always found that the mastoid could have been opened sooner with advantage to the patient. In no case did the finding justify further postponement. If we recall the indisputable fact that more than one-half of all brain abscesses, and more than one-half of all thrombotic diseases of all the sinuses of the brain are of otitic origin, we cannot fail to see that there is no exaggeration in the statement that intracranial disease is likely to result from a deferred operation.

In a service of thirty years in a general hospital where there were special otiatric and pediatric services, I had in the ear department at all times cases of suppurating ears in children. There were also cases of mastoid disease, cholesteatoma of the temporal bone, otitic serous and purulent meningitis, cerebral and cerebellar abscess, and thrombosis of the sigmoid sinus. These cases were recruited in large measure from the dispensary service, which directed the stream of cases to the various departments. I was also called to examine the ears of children in the pediatric service whenever they presented striking ear-symptoms. The number of these cases was small. If we remember that in this, as in other general hospitals, cases of measles, scarlatina, diphtheria, and other diseases of a similar nature are not admitted, we can understand why aural affections, so frequently accompanying the diseases mentioned, but rarely find their way into the pediatric service of a general hospital.

The foregoing facts largely explain the attitude of the pediatrist in private practice, especially in his relation to the otologist. The lack of aural material in his wards will lead him to believe that purulent middle ear disease, with its dire consequences, is of rare occurrence. The pathologist, however, tells us that ear disease in babies and children is very frequent, but remains unrecognized when there are no aural manifestations. Ponfick, for instance, (Berliner Klinische Wochenschrift, 1897,) found purulent otitis media, with or without other diseases, in ninety-one cases out of one hundred autopsies performed on material derived from the children's clinic in Breslau; cases in which the diagnosis had been made of broncho-pneumonia and gastro-intestinal catarrh. In the great majority of these cases the existence of the purulent disease of the middle ear was not suspected by the clinician.

The ear diseases which announce their arrival with great pain, high fever, perforation of the drum, are readily recognized and cared for; while the insidious cases in which the pus remains in the tympanic cavity, and either acts through

the blood or infects the respiratory or gastro-intestinal tract, remain unrecognized, and therefore do infinitely more harm.

I might say at this point that it is not an easy matter to examine the ear of a child, and when von Pirquet says that in cases of scarlet fever the drum-head should be examined without a speculum in order not to annoy the child, it is hard to see what knowledge we can derive from such a makeshift examination. The canal leading to the drum is often a very narrow tube, at times obliterated by fine hairs, wax, or scales, which have to be pushed carefully aside before the drum-head and its landmarks can be viewed. I consider an otoscopic examination in a child more difficult than an ophthalmoscopic examination, yet we see that men of insufficient otological training will perform paracentesis of the drum without previous accurate interpretation of the tympanic picture. The dab at the drum very often results merely in cutting the canal.

The great service we have yet to render to childhood is to recognize ear disease when there are no striking manifestations. This result will be obtained when the knowledge of the pathologist is applied by the clinician, and the ears of all sick children are frequently and thoroughly examined as a matter of routine.

TEMPERATURE CHANGES IN INFANTS AS AN INDICATION FOR THE MASTOID OPERATION.

By FRED WHITING, M. D., New York, N. Y.

The faculty of infants and small children for exhibiting sudden and extreme exacerbations of temperature upon slight provocation has long been one of the well recognized privileges of childhood, and when not associated with an actual or impending surgical condition has come to be regarded by the medical adviser with a considerable degree of complacency. This mental attitude of the doctor has gradually exerted a distinctly educational and disciplinary influence upon parents of the more intelligent class, until, in the present year of grace, we find that notwithstanding most mothers have a well-developed and pernicious thermometer habit, they are more amenable to reason and less liable to become hysterical upon finding a sudden and high temperature present in a sick child than parents of a similar social status were, but a few years ago, wont to be. Much of the credit for the institution and successful prosecution of this crusade of parental enlightenment must be attributed to the observation and teaching of the pediatricians, who, since the establishment of babies' wards in all the large hospitals, have enjoyed unrivalled opportunities for the critical study of the diseases of children collectively and in large numbers, and under circumstances most favorable to a just estimation and determination of their symptomatic value.

With large numbers of beds at their disposal, provided with adequate nursing and equipment, and unhampered by well-intentioned but misdirected and disconcerting parental influences, the pediatricians have been able to observe the course of acute diseases in an unprejudiced and scientific manner. The result of such experience has undoubtedly been to minimize their respect for temperature manifesta-

tions when unattended by other significant evidences of systemic disturbance. Indeed, to such an extent has wide experience justified them in this attitude that they have come to regard high temperature in infants as by no means convincing evidence of any serious condition, and we must be sufficiently magnanimous to acknowledge, however reluctantly, that the efforts of these gentlemen have to a certain extent modified our former embarrassment at the peremptory and insistent demands of patients for an immediate interpretation of the precise significance of each exacerbation of temperature. At the present day the surgeon's thermometer is as much a part of the regular equipment of a well ordered nursery as is the baby's bottle, and contributes in no small degree to the needless apprehensions of the mother and to the inconvenience and annoyance of the doctor. But it is useless to complain, for the domestic use of the thermometer has come to stay. Only let us pray that the day of the domestic miscroscope and culture tube may long be delayed.

We have all of us had opportunity to observe many cases of illness in infants in the course of which sustained high temperatures of unaccountable origin have vexed and confused our diagnostic skill, but have not caused any great anxiety for the reason that they were unattended by any recognizable focus of suppuration or by any well-defined and alarming physical signs indicative of disease of vital organs. Such cases, in large numbers, pass on to recovery and are characterized as grippe, or are designated by some similarly uncompromising term, and we are never the wiser for our observation of the case. The diagnosis is not made, but no alarm is felt at any time, because of the absence of focal symptoms. But should it happen by any chance that a patient exhibiting such temperature phenomena is suffering from acute suppurative ear disease, we are at once disposed to assume that the disquieting manifestations are all attributable to the purulent process and to ascribe to the symptoms a gravity of significance which greater experience demonstrates to be unwarranted.

A not infrequent experience which the babies' wards in the hospital furnish, is an opportunity to observe two infants occupying, perhaps, adjoining beds and exhibiting nearly similar temperature manifestations,—either continuously high ranges or rapid and perhaps excessive fluctuations. Neither case exhibits on physical examination signs indicative of serious involvement of any vital organ. Blood counts of the two patients, showing moderately high leucocytosis with a reasonably high polynuclear count, throw no especial light upon their conditions. But while the one case, having no recognizable focus of suppuration, is regarded with interested speculation only as to the cause of the temperature, and without further concern, the other patient, having a suppurative otitis media, is immediately invested in our estimation with a degree of importance to which in the majority of instances the subsequent course of the illness demonstrates it not to have been entitled. We find difficulty in convincing ourselves that a sick infant may have a suppurating ear which is nothing more than an incident of its illness. *We otologists are prone to take the view that an inflamed ear when attached to a sick infant must be responsible for all evidences of illness which the child presents.*

There is a reasonable excuse for the otologists thus laying undue stress upon the probable consequences of suppuration of the ear, for we have all of us had opportunity to observe similar conditions occasion the most serious and rapidly fatal results; and when a doctor has once experienced such an unfortunate outcome in consequence of attempts at conservative treatment he cannot divest his mind of the constant fear that such a deplorable experience is likely to repeat itself. Moreover, he knows that the dangers to be apprehended from an early mastoid operation in competent hands are few indeed, and he is perhaps not to be blamed if he prefers to expose the patient to the discomforts of an unnecessary operation rather than to assume the responsibility of the remote risk with which all delays in operating must inevitably be attended.

It requires the most careful weighing of conditions and

adjustment of balances to enable us properly to differentiate between the relative unimportance of a set of symptoms which appear in a given case unattended by suppuration and the precise value of similar manifestations when exhibited in another case in which suppuration is present. It is evident that we can hardly over-estimate the value of the symptoms if they are due to sepsis, and it is equally certain that we can afford to disregard them if they are not of septic origin.

An experience which the recent winter has several times furnished in the practice of the writer has been to be called upon to attend an infant which in the course of grippe has developed otitis media. Under the influence of this double infection, a very high temperature has developed which, notwithstanding a free myringotomy and evacuation of pus, has persisted for some days, in certain instances, with no considerable remission, and in other instances with very decided daily fluctuations, to the great anxiety of the family and concern of the doctor. Many such cases have rewarded a patient and conservative course of temporizing by gradual reduction of temperature and complete recovery without operation. In fact, so frequently have I experienced this result during the last year that I no longer feel disappointed when upon the performance of myringotomy in the presence of grippe there is not an immediate and pronounced reduction of temperature. Grippe infections require some days for their elimination from the system, and although the paracentesis of the membrana tympani has relieved pressure and established drainage, thus accomplishing all that we may properly expect from it, it cannot, at the same time, eliminate grippe infection. This is a matter of days. The question, then, narrows itself down to a consideration of temperature, and we may propound the question of whether or not we are justified in advising mastoid operation during an acute otitis media in which, without other assignable cause, the temperature remains high, or in which rapid and considerable fluctuations of temperature are present with-

out the usual local inflammatory evidences of the tympanum and mastoid to guide us.

We must bear in mind the fact that infants and small children exhibit high temperatures upon the slightest provocation, and that a condition which in an adult would cause only a slight elevation of temperature,—101° perhaps, or less,—and to which we should attribute no significance, will in an infant cause a temperature elevation quite out of proportion to the actual gravity of the condition, but is apt to be regarded with the same degree of consideration as a similar high temperature in an adult. Any such estimate of temperature values is based upon faulty observation, and greater experience will, I believe, tend to correct such errors. It seems to the writer that the same rules governing the appearance of local inflammatory evidences in adults may, with equal safety, be insisted upon in infants.

In the early years of life the bony structures are very soft and unresisting, and pus in the mastoid is apt to demonstrate its presence by early evidences of mastoid periostitis, redness, swelling, oedema, and, in a short time, by subperiosteal accumulations of pus. In fact, a daily widening experience with a large children's service in a hospital, is converting the writer to the view that in infants and children we may safely wait for the same classical signs at the fundus of the ear, namely, swelling and bulging of the drum, with sagging of the membranous portion of the meatus, and at least convincing evidence of tenderness of the mastoid region before proceeding to operation. It is a frequent experience in all large clinics to have children brought for treatment with red and swollen mastoids and with large subperiosteal accumulations of pus,—the so-called neglected cases upon which home remedies have been exhausted. Still, we find that such cases, as a rule, do well upon operation. They represent the extreme results of expectant treatment, home carelessness or neglect,—call it what you will,—and there is no reason to suppose that any great number of such neglected cases die at home and are not seen by doctors; wherefore it would appear that, at the worst, all that is

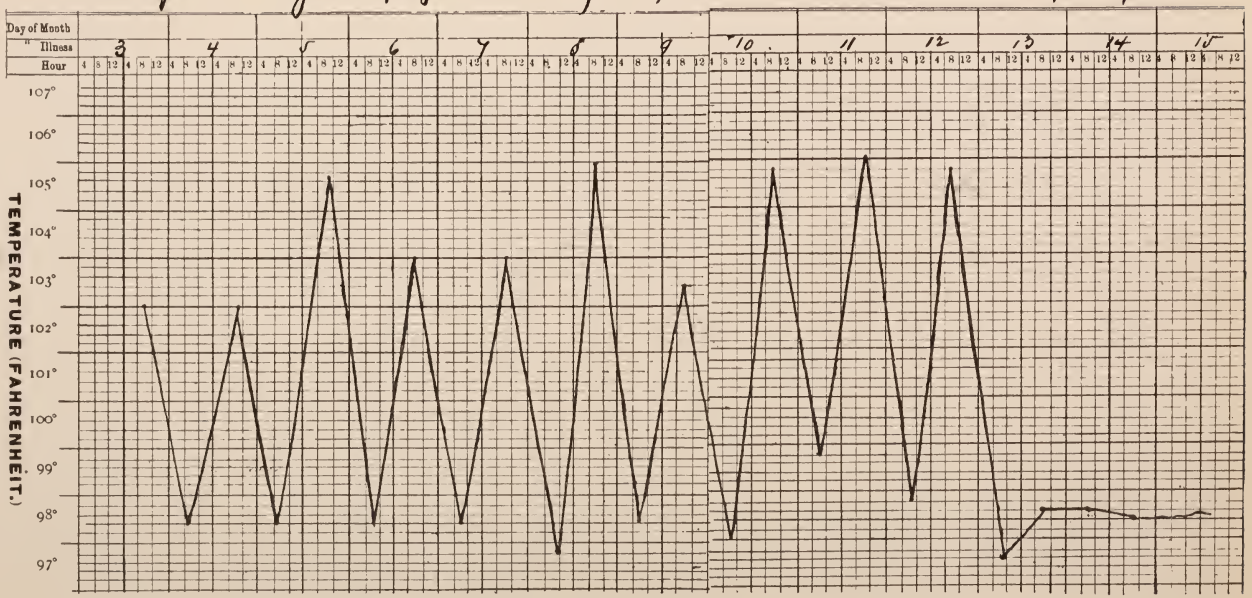
likely to happen from waiting for convincing signs of the presence of mastoiditis is a subperiosteal abscess, of which brain complications with very young children are rare.

It is not my intention to burden you with a long series of histories, although many typical cases have fallen under my observation. I shall content myself with reading one brief history and exhibiting one temperature chart.

The chart shows the temperature changes exhibited during ten days by a child one year of age which was under the care of a well known pediatrician of New York, who informed me that he could find no physical signs which might account for the temperature; he had made a diagnosis of grippe and asked me to examine the ears, suspecting them to be the probable source of the startling temperature picture here presented. Upon inspection, I found that both drums were bulging, and myringotomy was performed, evacuating purulent fluid from each tympanum. There followed for ten days one of the most startling pictures of septico-pyemic fluctuations of temperature that it has ever been my experience to observe, and the chart is here offered in evidence. It shows daily variations of from six to eight degrees for ten days, during which period the child at no time appeared particularly ill, and, when her temperature was low, would sit up in bed, play with her toys, take nourishment, and evince her usual interest in childish matters. With the onset of the high temperature, she became irritable and drowsy, but at no time had she a septic appearance. A copious discharge appeared from each ear, the drainage was apparently perfect, and myringotomy was not repeated. A smear made from the discharge showed the infection to be streptococcus. No blood count was made.

With the subsidence of the high temperatures, she made an uneventful recovery. At the time the observation was made, blood cultures had not been resorted to, and only the confident trust reposed by the parents in the assurances of the doctors that the child was not in danger prevented the performance of a needless double mastoid operation. Had the parents been of the nervous and apprehensive kind we

NAME Infant one year of age showing maximum and minimum daily temperatures



could not have controlled them and would have been obliged to operate.

When a focus of suppuration exists which we can clearly see,—as, for instance, a suppurating ear with the capabilities of which for producing serious trouble we are all well informed,—it is difficult for us to ascribe manifestations which may accompany such suppuration to the possible existence of other causes which we not only cannot see, but cannot convincingly demonstrate to be present. None the less, it involves no great tax upon the memory of any of us to recall one or more cases in which we were strongly disposed to operate on the mastoid, notwithstanding the absence of many of the classical symptoms, because of the persistent high or rapidly changing temperature, but upon summoning sufficient courage to wait a little longer the solution of the difficulty was suddenly reached in the discovery of a central pneumonia, a pyelitis, an adenitis, one of the exanthematous diseases, erysipelas, or some obscure gastro-intestinal condition. The causes of temperature are so numerous in children that we must be ever alert for their detection, lest some unsuspected agency contribute to our lasting mortification.

I know of no experience more valuable to an otologist than a close association with a large children's service in a hospital, for he there learns to interpret the significance of suppurative ear diseases not only from the somewhat radical standpoint of the ear surgeon, but also from the ultra- conservative point of view of the pediatrician. The pediatrician, with insufficient ear experience, is likely to be too conservative; the otologist, with insufficient experience in children's diseases, is equally prone to be too radical in method and hasty in judgment. The pediatrician sees everything from the viewpoint of the disease as it affects the majority of children, and does not allow sufficiently for the vagaries of the disease; the otologist, in his lack of experience, is apt to apply the same significance to high and fluctuating temperatures in infants that he would ascribe to the same temperature in adults. The pediatrician knows that a very slight disturbance will send up a child's tem-

perature, and until he can see the exact cause he refuses to believe that the condition is important. His conservatism is a good balance wheel on the radicalism of the otologist, and a combination of the two experiences is most valuable. The writer believes that the two classes of practitioners are gradually drawing nearer together in their estimate of the values of symptoms presented by infants, and are becoming less belligerent and more respectful to each other.

The otologist cannot forget that unfortunate case upon which he once operated because of unexplained and persistent high temperature and in which he encountered normal mastoid structures, while the central pneumonia which had been the cause of all the disquieting symptoms obligingly revealed itself on the following day,—and the derisive smile and patronizing air with which the pediatrician on every possible occasion reminds him of his blunder causes his sin of commission to linger long in his memory. As a parallel experience, the pediatrician has a vivid recollection of the jugular bulb thrombosis which terminated fatally through his insistent and fatuous dilatory measures, in one of his most important patients,—a sin of omission which the otologist would be quite too magnanimous in spirit ever to recall to his mind. But such experiences, emphasizing to us as they do the fallibility of our diagnostic acumen, are teaching valuable lessons of mutual dependence and respect, and will, I trust, in the near future, result in establishing a unanimity of opinion regarding the relative values of more or less constant symptomatic phenomena over the interpretation of which we are at present widely at variance. In the treatment of ear diseases in children the otologist and the pediatrician should be complementary to one another,—neither is sufficient to himself,— together, they make a strong proposition.

To what extent may the blood count in infants and young children be relied upon as a determining factor in deciding us to operate in cases where the only apparent urgent symptom is temperature? The count will frequently exhibit the most startling total and that, too, when the symptoms

present are not calculated to arouse any great anxiety as to the patient's welfare. Leucocyte counts of 40,000 to 50,000 are not uncommon in certain diseases from which children speedily recover, and Dr. Holt of New York, in consultation, recently cited a case he had just seen, in which the count showed 120,000 leucocytes, and yet the child did not appear very ill and recovered without even the aid of a diagnosis. The wide variations of the total count from day to day during the course of an ear inflammation when associated with some attendant disease, are at times very perplexing and disappointing, so much so as often to compel us to disregard them entirely. They offer their greatest diagnostic assistance in those instances in which, when repeated at brief intervals, they show a numerical regularity or a disposition to a steady ratio of increase. The utmost dependence that can be placed upon the count under the foregoing conditions is to lend a certain degree of corroboration to other strongly indicative signs when present; but under no circumstances can the blood count be relied upon to ratify a diagnosis of mastoiditis, the only other evidence of which is high temperature.

With the introduction and perfecting of blood culture methods, we have gained a valuable diagnostic aid in determining the significance of rapid and excessive fluctuations of temperature, for while a negative culture does not prove that no infection is present in the sinus, it is at least strong presumptive evidence of the absence of bacteriaemia, and an experience of eighteen months with a large number of suspected cases of sinus phlebitis has exhibited so few failures and so many successes of the method that I now place much reliance upon it when performed by men of sufficient experience. The technique of blood culture in infants and small children presents certain considerable difficulties, but they are by no means unsurmountable, and the diagnostic value of the procedure has, it seems to me, been established beyond all reasonable doubt. In any event, a negative culture is very reassuring to me when I am treating a patient with a suppurating ear who exhibits wide

fluctuations of temperature unaccompanied by physical signs of mastoid disease.

Far be it from me to advocate delay in operating for mastoiditis in children after convincing signs of the disease have manifested themselves at the fundus of the ear; but equally to be deprecated should be the attitude of the surgeon who with injudicious haste proceeds to operation simply because high temperature is present and it cannot be demonstrated that mastoiditis does not exist. To operate for mastoiditis in infants upon temperature manifestations supported only by blood counts and without corroborative evidence in the form of inflammatory changes at the fundus of the ear, displays not only a lack of conservatism, but a selfish disregard for the welfare of the patient.

Discussion.

Dr. Charles G. Kerley, of New York city, said that the recognized failure of clinicians to discover acute ear diseases in children and the great harm which may thereby result, reverted back to the otologist and the medical school. Undergraduate instruction in otology, the speaker stated, was very defective. Every year he examines men for hospital appointments whose knowledge of the subject is of the most casual nature.

The available literature on this subject previous to the past few years was misleading. The advances in otology as applied to pediatrics, had been made during the past five or six years.

When he prepared the article to which Dr. Gruening referred a very keen search through the literature failed to reveal anything which gave very much data concerning the symptomatology and treatment of aural diseases or suspected aural disease in children. Most of the data found related to the symptom of pain. The otologist taught that pain, as evidenced by tugging at the ear, head-boring, crying, tenderness on manipulation, was one of the earliest

symptoms of otitis in children. This is absolutely wrong. In a series of 72 cases reported in the article referred to, 69 per cent. showed absence of this element, pain, which is considered so important. Since that time he had seen at least 200 cases, and the percentage holds good to the present time. The younger the child the less evidence is there of pain and the less frequently is pain encountered. In 20 cases he had noted restlessness, which was relieved when the ear was opened. Pain is a symptom of very little significance from the diagnostic standpoint. He had called attention to this matter in his book, emphasizing the necessity for the early examination of the ears of children, and stating that otitis media is more frequently overlooked in children than any other disease. He agreed with Dr. Gruening that the pediatrist should be as familiar with otoscopic examination as with any other method of examination. The symptom which he had found most constant in otitic disease in children is fever. Many elevations in temperature that were otherwise not to be explained, proved upon bacteriological examination following paracentesis, to be of aural origin. Men should be taught to recognize otitis and how to open the drum membrane, (a simple operation), and that great harm may result, mastoid and sinus involvement, from this not being done. The matter of differential diagnosis in suspected aural conditions should be emphasized. The otologist perhaps, places too much reliance upon the symptom of temperature elevation. Temperature in the child is a very unreliable sign to base a diagnosis upon and should be considered in connection with other symptoms. Grippe and pyelitis may be factors in high temperature without any objective sign. Because a child has a discharging ear and fever, it does not mean that the ear or the adjunct structures are necessarily the sole occasion of the fever. At the same time a case proven negative with the exception of the infected ear with continuous fever might warrant mastoid exploration, even in the absence of prolapse and other canal changes. A symptom of much value in the diagnosis of mastoid disease rested upon the profuseness of the dis-

charge. The cases with very profuse discharge and with a persistent widely varying temperature often means mastoiditis. He had seen with Dr. Whiting such a case recover without operation. The condition of the child being critical to the degree of making the operation more dangerous than the disease.

As to mastoid tenderness, crying may be elicited from any child by sufficient pressure over the mastoid. This sign may be of importance and again it may be of very little value.

He did not agree with Dr. Gruening with reference to the lack of material for observation, as the children's hospitals, and the children's wards of general hospitals furnish ample material. The importance of aural disease in children has been appreciated only within the last few years. The papers of Dr. McKernon and his own, published five years ago, had done much to direct the attention of the medical profession to the right diagnosis of otitis media in infants and young children as well as to call attention to the dangers of overlooking the otitis.

Dr. Linnaeus E. La Fetra, of New York city, was in hearty accord with the general tenor of Dr. Gruening's paper. The more ears are examined, the more often is it found that the tympanic cavity is the site of inflammation and the cause of fever and fretfulness. It is very common, in the course of coryza or bronchitis, to find the sheen on the ear drum to be dulled or that there is a little redness or fullness at the upper part of the membrane or along the handle of the hammer. As Jacobi has so well taught, an otherwise unexplained temperature in a child is most likely to be due to an otitis media. Otitis media is exceedingly common in children. Personally, he had more need of the aurist than of any other consultant. There was hardly a week, from February to May each year, that he did not have to call the aurist to his aid three of four times a week. Dr. Gruening was right in saying that the aurist can be, and is, of the greatest help to the pediatrist. In the past ten years the

speaker had seen just *seven* cases of otitis media that were *complicated*. He could not say how many cases of otitis he had seen, but these complicated ones represent not more than one or two per cent. of the total number of cases. Naturally, he attributed this small proportion of complicated otitis to routine examination of the ears, whether the child makes any indication of ear pain or not. It is to be emphasized that infants do not localize pain well; often appearing to have merely abdominal distress or being simply fretful or biting on the fingers with the gums. The early and repeated incision of the drum membrane, as the otologists have taught, saves most cases from any further operation.

As to the attitude of the pediatrist toward the larger operations, he thinks it largely due to two factors: First, the pediatrist sees otitis in its early stage and sees it get well in the vast majority of cases after simple incision, while the aurist sees in his hospital and consultation practice the neglected or severe cases that are apt to go on to serious complications. Naturally, the prognosis of each man reflects the back-ground of his own experiences. Each can learn from the other. In regard to the more serious operations he thinks it more important for the aurist to have definite local indications before performing them. Three times in the past two years he had with advantage persuaded the aural surgeon to abstain from performing the mastoid or sinus operation, which was urged because of the great fluctuations of temperature after paracentesis. In one instance the surgeon was sure that these variations of temperature must mean sinus involvement and mastoid suppuration, although he could not demonstrate it by any local signs. The blood count in these instances may tend to confirm the surgeon in his opinion, since high leucocyte and high polynuclear counts are common. Unless, however, the aural surgeon can be confident, from local signs, that the mastoid is involved, he ought not to operate. General symptoms and especially temperature fluctuations, are not a safe guide in children, particularly in influenza. In connection

with influenza cases, he passed around a chart of a patient fourteen months old, showing an extreme range of temperature fluctuation. If the child had had, as is very common in grippe, an otitis media, the temperature alone would certainly seem to indicate some serious ear complication. He had found that the more experienced aurists are inclined to disregard temperature as a reason for mastoid operation. The younger men are more apt to be radical. It involves much more courage to wait in such cases than to operate. As regards the operation, the position of the pediatrists might be stated, in his opinion, as follows: The temperature alone is an unsafe guide, high leucocyte count with relatively high polynuclear is not a sufficient indication, particularly in cases of grippe. Persistent or recurrent discharge when not accompanied by sagging of the posterior wall or by local signs of mastoiditis is not sufficient. The aurist should be sure of his local signs before exploring the mastoid.

To the pediatrist no stethoscope is complete that does not have an ear speculum attached. Of the two instruments there is more real need by the children's specialist of the ear speculum. He can make a diagnosis of pneumonia or bronchitis without a stethoscope, but he cannot know what is going on in the ears without a speculum.

Dr. Edward B. Dench, of New York city, recalled having seen with Dr. La Fetra, the case in which sinus thrombosis was suggested. This was merely mentioned by him as a possibility, but he would not have operated. A single rise of temperature, with a polymorphonuclear count of seventy-nine per cent. was not sufficient indication for operation. The chart was interesting and the case itself was interesting. In his textbook he had mentioned the temperature, boring at the ears, and other symptoms, and he had also called attention to the fact that the temperature may be due to other causes, but that in cases of unexplained temperature the ears should always be examined. The instruction given in medical colleges at the present time is a great deal better

than that of a few years ago. The simple and acute diseases of the ear are being taught; the chronic diseases can be learned later. Otoscopic appearances in these cases are not always characteristic. Every year he sees cases in which redness of the drum membrane is not a prominent symptom, even where the disease has lasted for some time. He cited a case which he had seen with Dr. Kerley, in which the drum membrane was so little red that it did not seem to offer an explanation of the temperature fluctuations, and yet upon incision pus was found. There may be very little displacement of the membrane by the fluid, and the actual condition is apt to be overlooked. The mortality rate in infants upon whom the mastoid operation has been performed is not very high according to the speaker's experience. At the New York Eye and Ear Infirmary he sees a great many cases of mastoiditis in infancy, all severe cases, with the symptoms well established. He sees on his service perhaps as many as fifty such cases within a year, not more than one or two of which are lost. At one of the general hospitals with which he is connected he is very often asked to perform the mastoid operation as a conservative measure. He agreed with Dr. Kerley concerning the eliciting of tenderness in children. Most children will cry if sufficient pressure is exerted over the mastoid. The fact is often overlooked, however, that acute otitis, uncomplicated, in children tends to clear up. At the end of a week or two the discharge clears up, the muco-purulent discharge becoming less purulent and less profuse. If it continues for a number of weeks there is some trouble beyond the middle ear. He questioned whether it would not be more conservative surgery, where the discharge continues, to operate before the disease extends to the ossicular chain. At Saint Luke's Hospital not long ago a double incision was performed, the discharge persisted, and this was the only symptom except that the child was poorly nourished. After about two months it was decided to do a mastoid operation, when extensive caries was found. The child then began to improve very rapidly. The mastoid operation should be performed not for the

acute symptoms, but for the chronic condition, which must result in some impairment of hearing. The blood count in his hands had always seemed unreliable in these cases. The differential count is more important than the leucocyte count. One must depend upon the general symptoms of the case, together with the blood count. He had never opened the mastoid of an infant and found it normal.

Dr. George L. Richards, of Fall River, Mass., asked if there is any relation between acute pyelitis and otitis media, citing a case which suggested that there might be some such connection.

Dr. Arthur B. Duel, of New York City, said that soon after the appearance of Dr. McKernon's classical paper on "Primary Jugular Bulb Thrombosis in Children, as a Complication of Acute Purulent Otitis Media," he was appointed otologist to the Babies' Hospital. He was filled with the ideas presented in the paper. On observing several infants in whom wide vascillations of temperature took place, and who also had suppurating ears, he felt it his duty to advise exploration of the jugular bulb. Dr. Holt did not concur with this opinion. At first he regretted this, but as time passed and he saw one after another of the children recover without operation he was pleased. In one case he had the opportunity of seeing an autopsy on the child who had died of pneumonia, and greatly to his surprise he found the bulb and sinus clear. Shortly after that he read a paper in which he called attention to many of the points mentioned in Dr. Whiting's paper; also stating that it was impossible to conclude, on temperature alone, that an operation was indicated on the sinus or bulb in children under two years of age. The mortality in these young infants after operation is considerably higher than in adults. Success in operating upon young children has been largely increased and the mortality lessened since surgeons have been more careful in the control of hemorrhage. Loss of blood is often the cause of the fatality in the operations under discussion.

Attention was called to the difference in the types of children found in the special hospitals and in those like the 'Babies'. The mortality has been greatest in patients known as "feeding" cases, where assimilation is bad and vitality low. Such patients do not die as the result of operation *per se,* but because of this injury added to the already serious condition. They would undoubtedly die without operation, but death is hastened by this added shock. The otologist who operates upon an infant under two years of age needs the pediatrist to help him in the subsequent care of the case just as much or more than the pediatrist needs him to do the operation. At the Manhattan Eye and Ear Hospital a pediatrist takes care of the subsequent feeding of infants after operation.

Dr. James F. McKernon, referring to the statement that one should not operate upon the symptom of fever alone, said that after all diseases have been eliminated by the general practitioner and the pediatrist, one must come back to the original focus for an explanation of the temperature. The ear will furnish the explanation. On the other hand, a high temperature of septic origin is indication enough for operation. He concurred in what had been said concerning temperature alone, but emphasized the fact that there are exceptions, and that these occur very frequently. He cited a case of streptococcic infection, which was seen by Dr. Whiting, in which there was absolutely no indication of the disease, from the physical standpoint, except the temperature, which went to 105°. There was no change in the fundus, no mastoid tenderness, and yet the mastoid was necrosed from one end to the other. The temperature fell, then rose again, and the other ear was found to be involved. This showed the classical symptoms which are associated with involvement of the mastoid. With high temperature, without the classical physical signs, when other diseases have been eliminated, one must come back to the ear to account for the symptom.

Dr. Frederick L. Jack, of Boston, cited a case he had recently seen in consultation in which repeated high temperature was absolutely the only indication of mastoid involvement. The mastoid operation was advised, and the diagnosis verified.

Dr. B. Alexander Randall, of Philadelphia, emphasized the fact that these acute cases have a strong tendency to the formation of non-septic thrombus and the closing off thereby of the jugular as completely and safely as it can be done by ligation. In many such cases where autopsy is made, the jugular is found to be entirely closed. Even the cavernous sinus may be thus involved, as in a case which he reported some years ago, and its lumen re-established or collaterally substituted later.

Dr. Gruening, in closing the discussion, said the statement attributed to von Pirquet, which Dr. Kerley questioned, viz., that in scarlet fever the ear of the child should not be examined with the speculum and could be examined by merely drawing the ear back, because the introduction of the speculum causes annoyance to the child, is found in the article on scarlet fever in Pfaundler and Schlossmann.

With reference to that imponderable something called intuition, which the pediatrist attributes to the otologist, he said many things are involved. First of all, the amount of pus, mentioned by Dr. Dench, forms such an indication. This may be wiped away and immediately it wells up again. It cannot come from the middle ear; there must be a cavity from behind which furnishes this pus. It is not necessary that there be visible changes in the canal, but this, in connection with the temperature, would be indication for operation. This cannot be communicated, but belongs to the *imponderabilia* of which the pediatrist speaks. Without tenderness, without pain, without temperature, the otologist sometimes recognizes such cases and knows that there must be mastoid disease where there is absolutely no text-book symptom to guide him. These cases are often met with, and

it is very important that the pediatrist should become acquainted with them. It is not easy to get the tympanic picture. Otology should not be taught by lectures, but at the bedside or in the clinic, where the student should be taught to inspect the drum membrane of the adult and of the child.

Dr. Whiting, in closing the discussion, said that at the present time otology is taught in one institution with which he is familiar, and probably in others, after the method upon which Dr. Gruening insists. While it is taught didactically to a certain extent, the students are also required, in small sections, to examine ears. Notwithstanding the views expressed by some of the speakers, temperature alone seldom guides one to operate. He mentioned in this connection a case seen in consultation with Dr. Kerley. The amount of discharge from the ear was tremendous, and the temperature exacerbations were such as to cause the greatest concern, but the child's condition was such as to preclude operation, and it made a perfect recovery. Dr. McKernon's contention is quite true, occasionally one encounters a case which goes contrary to all established laws, and with nothing but temperature to guide us we must operate. But such cases usually look sick and show prostration out of proportion to the other manifestations of the disease.

OBSERVATIONS ON THE ASSOCIATION OF SUPPURATIVE DISEASE OF THE NASAL ACCESSORY SINUSES IN ADULTS AND ACUTE OTITIS MEDIA.

By CORNELIUS G. COAKLEY, M. D., New York.

The subject indicated in the title of this paper is one to which the writer has given considerable attention for several years.

The clinical study has been almost entirely on patients seen in private practice, thus affording an opportunity for a more thorough examination than can usually be given to the individual patient in a clinic.

While many of the cases of suppurative nasal accessory sinus disease are easily diagnosed, others are only recognized after cleansing the nares, thoroughly contracting the nasal mucous membrane by cocaine and adrenalin, and observing after a lapse of a quarter of an hour or more, the escape of secretion into the nares or naso-pharynx.

Transillumination, in the case of the antra, and to a less extent for the frontals, is an important aid to diagnosis.

We have chosen to limit our report to adult cases, because, while suppuration in the nasal accessory sinuses undoubtedly occurs in children from the time of the earliest appearance of the sinuses up to the time of their full development, it is often very difficult to be positive of the diagnosis of sinus disease.

The factors which in children prevent our satisfactory examination are:

First: The small size of the nasal chambers.

Second: The proportionately larger size of the turbinal bodies.

Third: The relatively greater amount of swelling of the turbinal bodies when acutely inflamed.

Fourth: The intractability of children to the necessary application of cocaine, and the use of instruments for probing and puncturing the cavities.

Fifth: Transillumination in most children under twelve is unreliable or positively misleading. The two antra develop so unevenly that one may be much darker than the other, and yet, if the darker one is washed out, no secretion is found in it.

It seems best to divide the subject into two parts: (A) Statistics, and (B) Personal impressions.

(A) Statistics.

We have analyzed the histories of all *new* patients seen between October first, 1909, and March first, 1910, who came to us suffering from

Acute suppurative otitis media,
Acute rhinitis,
Acute sinusitis (using this term to indicate suppuration of one or more of the nasal accessory sinuses), and
Chronic sinusitis.

There were 16 cases of Acute suppurative otitis media,

There were 26 cases of Acute rhinitis, without sinus involvement,

There were 31 cases of Acute sinusitis, (all these cases also had an acute rhinitis.)

There were 36 cases of Chronic sinusitis.

We shall study these cases from an Otological and a Rhinological standpoint.

Otological Standpoint.

The sixteen cases of acute suppurative otitis media sought relief of the earache, and regarded the accompanying "cold in the head" as of little moment. The examination of the fundus revealed a red and bulging membrana tympani. A spontaneous perforation had occurred in three cases. In all sixteen the operation of myringotomy was performed usually after inducing nitrous oxide anesthesia.

In three of the sixteen cases, nineteen per cent., a thorough examination of the nares and sinuses showed only an acute rhinitis, with no involvement of any of the sinuses. All three made an uneventful recovery.

Of the thirteen remaining cases, all had an acute rhinitis complicated by acute sinus disease; that is, eighty-one per cent. of this series of acute otitis media had at the time of their first visit, and antedating the onset of the aural disease, a suppuration in the nasal accessory sinuses.

Of these cases, eight made an uneventful recovery for ears and sinus disease, while in five, or thirty-nine per cent., the disease extended to and involved the mastoid to such an extent that a mastoid operation was performed.

In one case, a nurse, Miss C....., had acute rhinitis, complicated by right antrum, four days before pain in ear began. The pain in ear commenced at six p. m., at eight-thirty p. m., nitrous oxide anesthesia and myringotomy. Smear at time of operation, pneumococcus. Second day, profuse bloody discharge and mastoid tenderness; third day, mastoid tenderness increased with considerable prolapse of posterior-superior wall; fourth day, tenderness of mastoid increased with edema of skin over mastoid, prolapse more marked.

As there were no facilities for operating on the mastoid or caring for the patient in the Nurses' Home, she was sent to the New York Eye and Ear Infirmary, where Doctor E. B. Dench exenterated the mastoid, the patient making an excellent recovery.

One of the cases, a nurse, Miss B....., had influenza, diphtheria and an acute pansinusitis. Both mastoids were involved, and operated on by my associate, Doctor H. M. Taylor.

Another mastoid case, Mrs. R....., had suppuration in left frontal, ethmoid and antrum, probably also left sphenoid, but a septum enormously deflected to the left, prevented examination of the left sphenoid. Left mastoid and sinuses healed.

Miss S..... had right frontal, ethmoid, antrum and sphenoid. Operation, right mastoid and recovery.

Another case, Mr. M....., had right antrum alone. Operation, right mastoid and recovery.

A very interesting clinical fact in this series of mastoid cases is that the infection of the tympanum and mastoid was limited to the same side as that of the nasal accessory sinus disease. In the bilateral mastoid case there was a bilateral nasal involvement.

Of the eight uncomplicated cases of otitis media, *all* had pus in the antrum of the corresponding side. Three also had pus in the frontals of the same side and two had pansinusitis.

Three other cases sought relief from a severe earache. The pain was referred to the auditory canal and mastoid region. In two of the cases the pain was unilateral, in one bilateral.

In all of these cases the fundus and canal was normal. The slightest touch of the skin over the mastoid proved exquisitely tender. They were typical cases of otalgia. In one case the antrum on the painful side was filled with pus. A few irrigations of the antrum cured it and the otalgia. In the bilateral otalgia case there was a chronic pansinusitis. On establishing good drainage from the sinuses, the otalgia disappeared. The third case had a chronic suppuration in the sphenoid and posterior ethmoid of the otalgic side. She still has exacerbations and remissions of her sinus disease and pari passu of the otalgia.

Rhinological Standpoint.

There were twenty-six cases of Acute Rhinitis. In order that there may be no misunderstanding about our cases of acute rhinitis, I wish to state that it is unusual for me to see the milder forms of this disease. Most of the cases are accompanied by hypervascularization of the tissues and considerable hypersecretion, the character varying from watery to muco-purulent, according to the stage of the disease. It is often difficult to be certain that there is not some slight

involvement of the sinuses. If either antrum on transillumination was darker than the other it was irrigated.

Of these twenty-six cases, twenty-three did not have acute ear symptoms or signs other than a feeling of stuffiness from tubal congestion, and a slight dilatation of the vessels of the tympanic membrane.

Three of the twenty-six, or eleven and a half per cent., had acute suppurative otitis media. I have been unable to find in rhinological or otological literature any figures for comparison by which to judge whether this percentage of aural complication is high or low.

Acute Sinusitis.

There were thirty-one cases of acute sinusitis. Thirteen of these cases, or forty-two per cent., had an acute suppurative otitis media as a complication, at the time they came under observation.

The ears were treated as heretofore described. The treatment of the sinus disease consisted in contracting the nares with a two per cent. solution of cocaine, waiting for ten minutes or longer, until by inspection the swelling of the nasal mucous membrane had subsided, and gentle irrigation of each naris, by means of a syringe containing a warm (T 110 degrees F.) normal saline solution, to which 0.3 per cent. bicarbonate of soda was added. Further cocainization beneath the inferior turbinate, and irrigation of one or both antra through a Myles canula passed through the nasal wall beneath the inferior turbinate into the antrum. If the frontal sinus did not drain satisfactorily, the anterior end of the middle turbinate was amputated. The sphenoid was irrigated by means of a canula inserted into the cavity through the ostium.

Of the eighteen sinus cases, not complicated by otitis media, when first seen, there was but one case which subsequently developed an otitis media demanding operation.

H. S. S., Jr., had a slight intermittent earache for forty-eight hours, for which I was consulted. The right fundus was red with only a slight bulging of Schrapnell's mem-

brane, hearing slightly impaired. The left fundus was normal. He had a pansinusitis. I did not perform myringotomy, regarding it as a catarrhal condition which might resolve without operative interference. The nares were irrigated twice daily and the maxillary sinus daily or every second day for a couple of weeks. At the end of that time the fundus had nearly returned to normal, the hearing was normal and the acute rhinitis and sinuses practically well.

He was exposed to some fresh infection (possibly a mild scarlatina), his sinuses discharged worse than ever, and there was a pronounced redness and bulging of the right membrana tympani requiring a myringotomy, which was performed by Doctor McKernon.

In none of the other seventeen cases, although their nares were repeatedly carefully syringed, did any aural complication develop.

I believe that the aural complication in that one case was the result of the new infection, rather than any fault in the technique of irrigation.

In the thirteen cases associated with otitis media the treatment of the nasal sinuses was conducted as described for the uncomplicated cases.

Chronic Sinusitis.

There were thirty-six cases of chronic sinusitis. The duration of the disease varied from two years to twenty or more.

In four cases the disease was limited to one antrum; in one case to the sphenoid and posterior ethmoid of one side; in sixteen cases an unilateral involvement of all the sinuses, and in fifteen cases there was a bilateral involvement of all the sinuses.

Although all of these cases were under treatment by syringing, various intra-nasal operations, etcetera, for some weeks, but one case developed an acute otitis media.

That patient, Miss N....., had pansinusitis; she gave a history of having had an otitis media before coming to me. Both antra had been widely opened intranasally.

A date was set for an external operation on the frontals. On the morning of that day she came to the office with a spontaneous rupture of the right membrana tympani and a purulent discharge. The sinus operation was postponed until the otitis disappeared.

In this series, one case in thirty-six, about three per cent., developed an acute otitis media.

To recapitulate:

Eighty-one per cent. of acute otitis media cases had sinus disease,

Eleven and a half per cent. of acute rhinitis had acute suppurative otitis media,

Forty-two per cent. of acute sinusitis cases had acute suppurative otitis media,

Three per cent. of chronic sinusitis cases had acute suppurative otitis media.

(B) Personal Impressions.

The clinician gradually accumulates an experience which is invaluable in forming his judgment as to diagnosis and treatment.

While the above statistics cover too short a period, and too few a number of cases to be accepted as final, I wish to add some observations which I feel sure will be concurred in by other clinicians since attention is called to them:

First: Otologists agree that the infection in a large percentage of cases of acute otitis media proceeds through the Eustachian tube, complicating an acute rhinitis and acute naso-pharyngitis.

We believe that the severer types of acute rhinitis accompanied by acute infection of the nasal accessory sinuses are far more apt to be complicated by aural disease than the milder types of acute rhinitis.

Second: It is extremely rare for a patient with acute sinus disease, coming to us in the early stages, before acute otitis media has developed, to subsequently, during the course of the treatment, develop an acute otitis media.

Third: We are convinced that the early recognition of acute sinus disease, and appropriate treatment for its relief, will prevent many a patient from developing acute otitis media.

Fourth: The fact that acute otitis media usually occurs on the same side as the sinus disease inclines us to the belief that the pus from the various sinuses bathes the pharyngeal orifice of the Eustachian tube and is thence forced into and infects the tympanum.

Fifth: Cases of acute otitis media, associated with nasal accessory sinus disease, are more likely to develop such a degree of mastoiditis as to require a mastoid operation, than otitis cases not complicated by nasal accessory sinusitis.

One explanation of this may be in the more severe character of the infection. I am inclined to believe, however, that some patients would repair the damage to the tympanum and mastoid if relieved of the added burden of the nasal disease.

We have repeatedly seen cases of severe otitis media and mastoiditis, with all the indications for mastoid operation, recover without a mastoid operation when the nasal sinusitis was recognized and treated.

Sixth: Patients suffering from chronic suppuration in the nasal sinuses are much less prone to acute otitis media than those having acute sinusitis.

In the light of modern bacteriology this may be accounted for in two ways:

First: The bacteria found in the pus in many cases of chronic sinusitis grow very poorly on any nutrient media. They seem to have lost much of their virulency.

Should they be forced into the tympanum through the Eustachian tube, they may not infect the ear.

Second: It is also probable that the antibodies formed in the chronic cases are a prevention against inciting a new infection.

It is a well known fact that after any operation, intranasal or external, on a case of chronic sinusitis, the virulency of the bacteria is greatly increased owing to the wound

secretions forming an excellent nutrient medium; hence the cellulitis or erysipelas which develops after some of our external operations on cases of chronic nasal accessory sinus disease.

We are not surprised that a small percentage of patients having chronic sinusitis, on whom intra-nasal operations are performed, develop acute otitis media or even mastoiditis. The wonder is that more do not.

Discussion.

Dr. William H. Haskin, of New York City, agreed that cases of acute suppurative inflammation of the nasal accessory sinuses are frequently complicated with middle ear disease. During the last few years in the treatment of these cases he has rarely had this complication, and he attributed it to the fact that he does not syringe the nose. After applications of cocaine, wonderful relief is obtained in these cases by cleansing the sinuses with the vacuum cleaner once or twice a day at least. Since using this method he has had no ear complications.

Dr. George L. Richards, of Fall River, Mass., had had two cases of acute otitis media following frontal sinus disease. In one both ears were affected. From the amount of the discharge he thought the mastoid must be affected, but both cases recovered without mastoid operation. In one the ear trouble lasted longer than the sinus disease. In the other the pathologist found the bacillus catarrhalis, but neither streptococcus nor staphylococcus.

Dr. John E. Sheppard, of Brooklyn, mentioned a case of mastoiditis in the pus from which the pathologist found the bacillus catarrhalis. The prevention of ear complications from nasal sinus disease should be emphasized. Some of the causative factors are the ignorance on the part of the patient of the proper way of blowing the nose, the use of the nasal douche, and the use of the Politzer bag.

Dr. Fred Whiting, of New York City, thought the reader of the paper should say whether, in those cases of threatened mastoiditis which recovered under the treatment he suggested, the symptoms of mastoiditis were distinct enough to convince one of the necessity of the mastoid operation, or whether they were merely sufficient to make one fear that the mastoid is involved.

UNSUSPECTED MIDDLE EAR CONDITIONS FOUND IN ROUTINE POSTMORTEM EXAMINATIONS.

By J. E. WELCH, M. D., New York, N. Y.

Several years ago while in charge of a special service, in one of the large hospitals of New York, consisting of erysipelas patients, I was impressed by the number which after the disappearance of their symptoms of erysipelas developed multiple abscesses over the body and extremities and later middle ear and mastoid complications which required operation. The middle ear and mastoid complications were considered as metastatic foci and analogous to the surface abscesses which had their origin in a general streptococcaemia resulting from the organism of erysipelas. In the light of later findings of middle ear diseases associated with various other systemic infections, the point can now be raised as to whether in some of these cases we were not dealing with an erysipelas secondary to middle ear contamination.

The observation of associated lesions in this particular condition prompted a further and routine study of the middle ear content in all postmortem examinations, which now includes about two thousand autopsies, 2.050 deaths being due in these cases to a variety of causes.

Bacteriological examination of secretions obtained during life from the tympanic cavity, through spontaneous rupture wounds or paracentesis punctures, shows the presence most frequently of pneumococci, streptococci, staphylococci, a combination of these latter two, bacillus pyocyaneus, colon bacillus, influenza bacillus and the meningococcus. We would naturally expect the complicating systemic disease to be one caused by the same organism as that found in the middle ear discharge, and as a rule this is true.

A digression to the consideration of the minute anatomy of the middle ear would be out of place before this body of

surgeons, but there are certain points in this connection that must necessarily be prominent in mind in order to appreciate the pathological contents of this cavity, which we shall soon review.

The perverted physiological processes that act to produce pathological lesions produce varying effects depending upon the nature of the tissue involved. In general, exudation is apt to be limited in compact tissues like bone, more extensive in looser tissues like the lung, and very extensive in the body cavities where the support for the blood vessels from the surrounding tissues is slight. We have in the middle ear an irregular cavity lined by mucous membrane. The mucous membrane has nutrient vessels coursing through it. Its outer surface is supported by a resistent wall of bone, its inner is not supported and therefore any swelling of the membrane from congestion, oedema or exudation will cause an encroachment of the membrane upon the ear cavity to the full extent of its elasticity. Beyond this point the products of exudation or transudation discharge into the cavity of the ear and form the various accumulations to be found in this cavity. If the exudate be slight, later absorption is possible, if extensive, improbable. As is the case in certain other cavities drainage from this one is not ideal. The Eustachian tube communicates with it above the floor, leaving a basin for accumulation. The tube itself not infrequently has its lumen encroached upon by hyperplasia of the lymphadenoid tissue which is to be found in places beneath its epithelial lining. While the individual is in the erect attitude the tube is in a good drainage position, but about one-third of the time, or practically eight hours in twenty-four while he is in bed, it points upward giving still more opportunity for stasis in the middle ear. This is true in health, but in conditions that confine one to bed the tube is continuously in this almost vertical position.

In none of the cases forming the material of this report was the drum membrane ruptured. The various secretions and exudates were found only after opening into the ear, through the tegmen by chiseling it away. In every case the

membrane was subjected to internal inspection and found intact. The material found in the middle ear in no case was suspected antemortem. The findings fall naturally into the following classification.

Secretions—

Mucous.

Transudates—

Watery, amber colored, milky.

Exudates—

Serous, sero-sanguinous, gelatinous, purulent, muco purulent, fibrino-purulent.

Hemorrhages.

Mucous when found in the middle ear is an expression of a catarrhal condition caused usually by an organism which in smears appears as a diplococcus and in culture develops a streptococcus.

Transudates, as a rule, when found in adult ears, have associated with them an oedematous condition in other parts of the body, either in the subcutaneous tissues or in some of the body cavities or in the lungs. They are absolutely colorless or watery in appearance, or in instances where they contain some of the body pigments have a decided amber color. A milky looking transudate is frequently found which has an appearance very much resembling pus, but on microscopic examination is found to be composed of thin serum and mucous through which is an abundant admixture of epithelial cells but no pus cells. This type of transudate is common in the ears of children and is frequently called pus in the absence of microscopic examination. Various microorganisms may at times be found in these transudates, but as a rule they are ineffectual in producing inflammatory conditions.

The exudates are invariably caused by organisms and may be present as the sole lesion or may have associated with them a general systemic infection caused by the same pathogenic germs as those producing the middle ear exudate.

The most commonly encountered diseases in which middle ear exudates are found are streptococcaemia, staphylo-

coccaemia, lobar pneumonia, and cerebro spinal meningitis. In many instances the source of infection for any of the above named diseases is clearly evident. Doubtless every one of you have seen a bacteraemia appear after operation when drainage from the ear or mastoid was good. If under favorable conditions of drainage, a systemic infection may occur, how much more likely is it to occur where no such drainage exists? Fifty-seven per cent. of adults dying of streptococcaemia and thirty-three per cent. dying of staphylococcaemia showed a middle ear exudate containing the corresponding organism. In all of these cases the path of blood infection was definitely known not to be through the middle ear. In babies we find conditions different. In none of the cases was the route of blood infection known. Of those having streptococcaemia, thirty-three per cent. had a middle ear exudate containing the streptococcus, and of those having a staphylococcaemia, sixty-six per cent. had an exudate in the ear, showing staphylococci.

In the staphylococcaemic babies having the ear involvement, in which no other avenue of blood infection could be discovered, it seems reasonable to suppose the infection came through the ear. The staphylococcus is a very constant organism about the nipple and might easily be lodged in the mouth during nursing and be transmitted through the Eustachian tube to the ear and thence to the blood stream.

In two cases of multiple abscesses of both lungs in babies the infection seemed beyond a doubt traceable to the tympanic cavities. The middle ears were full of yellow pus which resembled that found in the lung abscesses and cultures from both sources developed a pure growth of staphylococcus aureus. A fact in proof of this theory is, as has been pointed out by Libman, I believe, that cultures from the spleen and other organs below the diaphragm develop no growth, showing the infection to be limited to the lesser circulation of the thorax. These cases are fulminating and die before the infection can spread through the general arterial circulation.

In ten per cent. of the cases dying of lobar pneumonia there was an exudate in the middle ear containing the pneumococcus. Studies of recent years have demonstrated that a large percentage of the individuals having this disease are subjects at the same time of pneumococcaemia. Since pneumonia has been put in the class with the septicaemias it is more than probable that many cases do not originate through the respiratory passage but by the organism passing by way of the middle ear into the circulation and finally lodging in the lung. The pneumococcus is found in the discharges from the middle ear during life far more frequently than any other organism and this would tend to strengthen the assumption just made.

As in the case of pneumonia there would seem to be a no less direct connection between the middle ear and the main lesion in epidemic cerebro spinal meningitis. In this condition twenty per cent. of the cases showed the tympanic cavity filled with a purulent exudate which contained the meningococcus. It is now generally accepted that the path of meningeal infection in this disease is by way of the nasal passages and especially through the cribriform plate of the ethmoid. The meningococcus is also found occasionally in the discharges from the ear, in view of which fact it would seem reasonable to assume that some cases of epidemic cerebro spinal meningitis originate from a primary middle ear infection, especially in the cases having no drainage through the drum membrane.

You will be spared lengthy history recitals, but I wish to put before you, very concisely, two records which illustrate a connection between unsuspected middle ear involvement and subsequent blood and meningeal infections.

The first case was that of a baby one year old having for about three days the symptoms of a cold in the head. Slight cough soon appeared, also vomiting and evidence of pain in the left hip joint. The physician summoned in the case called in immediate consultation a pediatrist and as a result of the conference a probable diagnosis of grip with gastro-intestinal disturbance and joint affection of some sort, was

made. Physical examination was negative with the exception of possible slight liver enlargement and signs of left hip arthritis. Treatment was carried out with the idea of controlling the gastro-intestinal disturbance by proper feeding, etc. Vomiting occurred only a few times during the next two weeks of the baby's life and was no more than might be expected from a disturbed alimentary tract. It exhibited no grave symptoms or sign of meningitis. Other joints became involved as follows: left wrist and elbow, right hand and possibly right hip. The temperature gradually rose to the neighbourhood of 104 degrees, toward the last of the disease, and the pulse to 160. During the last twenty-four hours of life there were observed muscular twitchings of the arms and occasionally the child would straighten out the legs. Death came suddenly on the afternoon of the twelfth day, consciousness having remained to the end. Inspection of the drum membrane was made several times, the last being on the day of death but no sign of middle ear exudation was detected. Autopsy was made about five hours after death, which revealed the following: all of the joints above mentioned with the addition of the left sterno-clavicular, contained a large amount of very thick, dry, gray colored pus. There was a slight bronchopneumonia of both lungs. The sulci over the entire surface of both cerebral hemispheres, the surface of the cerebellum and the pia at the base of the brain showed the same thick, dry, gray pus as that found in the joints. The right middle ear was found normal, the left was filled with pus like that above mentioned. Cultures from all of the pus foci and from the right heart and spleen all developed a pure growth of the influenza baccillus. Here the sequence of events would seem clearly outlined and appear first as an influenza cold with extension into the middle ear and subsequent infection of the meninges and blood stream with the formation of metasteses in the joints.

The second case was a male, porter, age forty, for three weeks had been having intense pain over the left side of

the head. On admission to the hospital had a few convulsions and lapsed into coma. Was operated, without anaesthesia, for cerebral abscess and two ounces of pus evacuated from the left temporo-sphenoidal lobe. Died twenty-four hours later. Post-mortem examination revealed the left middle ear and mastoid cells full of pus with the drum membrane intact. There was necrosis of bone just internal to the middle ear cavity and the dura just over the dead bone was elevated by about one dram of pus. There was a purulent exudate beneath the pia over the abscess area, and the cerebral ventricles were filled with turbid serum. Cultures from the pus developed streptococci and staphylococci. Here again the ear was not taken into account in the ante mortem consideration of the case, though it was quite surely the original focus of the trouble.

Hemorrhages have been found in the middle ears of those dying of eclampsia and hemophelia neonatorium. The majority of eclamptic women recover and with the use of human serum the bleeding babies also. It is probable that some of those who recover may have in one or both ears blood which may become infected by organisms and become a pus focus, therefore in recoveries from these conditions the middle ears should be carefully watched.

Forty per cent. of babies two months of age and under have one or both ears filled with material which can be placed in the above classification, and fifteen per cent. of adults also show the same. These being unsuspected conditions, and being entirely left out of the clinical consideration of the cases, point out the necessity both for more careful routine clinical work, and for some more satisfactory way of learning of the contents of the middle ear than by looking through the drum membrane only. Other blind cavities of the body have been brought nearer the surgeon's observation by means of various kinds of catheter so that now even the remote cavity of the kidney pelvis is no longer the mysterious hidden recess it was once considered to be. If it were practicable to use such an instrument through the Eustachian

tube, there is no doubt that better diagnosis could frequently be made, perhaps treatment altered and often life saved by diverting a fatal septicaemia or meningitis.

Discussion.

Dr. Joseph A. Kenefick, of New York City, emphasized the importance of the pathological side of conditions which are so familiar clinically. This is especially important in children. At the New York Foundling Hospital he had for years noted the connection between the middle ear cavity and pneumococcus infection. It is safe to say that of all deaths of children under two years of age a large number are caused by broncho-pneumonia, where the infection is endogenous from the middle ear cavity.

Dr. Wendell C. Phillips, of New York City, was impressed several years ago, at the time of an epidemic of cerebrospinal meningitis, with the number of instances in which the bacteriologist reported the presence of the meningococcus in the aural discharge from cases which recovered without developing meningitis.

Dr. George F. Fiske, of Chicago, inferred that Dr. Welch meant to convey the idea that finding pus in the tympanic cavity post-mortem would signify that it was present in life. Such is not necessarily the case.

SOME CASES OF OTITIC MENINGITIS.

By J. E. SHEPPARD, M. D., Brooklyn, N. Y.

CASE I. Eugene W., aged 29, male, German, a brewer, was admitted to my service in the Jewish Hospital May 26th, 1909, and came under the care of my Associate, Dr. Stickle, to whom I am indebted for the following history:

There had been a discharge from the left ear since childhood following scarlet fever, accompanied by more or less tinnitus and deafness. A week ago the discharge became somewhat bloody. The present illness dates back to five days ago, when he began to suffer from severe throbbing headache, at first frontal, and, spreading until it seemed to involve the entire head, became constant and intense. The following day there was added to the headache a marked degree of dizziness, but as yet no fever. The third day projectile vomiting developed. The fourth day there was some amelioration of all the symptoms, but on the fifth day he again began to vomit, had fever, headache returned with severity, and the neck began to feel stiff. There was no pain in the ear or behind it. On admission to the Hospital late on this, the fifth day of his illness, the patient was suffering from terrible headache, referred to the whole head, there was rigidity of the muscles of the neck and back, the head being much retracted, there were spastic seizures of the right arm and leg, a considerable degree of lateral nystagmus, Kernig's Sign present on both sides, tache cerebrâle well marked, reflexes were normal: most of the left tympanic membrane was gone, and the tympanic cavity was partially filled with granulations.

Examination of the eyes showed moderate dilatation of the pupils, which were equal, reacted rather sluggishly, and the eye-grounds were normal.

As a result of spinal puncture there were removed 40 cubic centimeters of opaque spinal fluid, which showed 95% of

polymorphonuclear cells, 4% of mono-nuclears, and 1% of endothelial cells, and from it there developed in the incubator the bacillus mucosus capsulatus, the same germ being found also in the material removed from the mastoid antrum, and in the intra-dural pus.

On May 27th, the day following his admission, a radical mastoid operation was performed; a relatively large necrotic destruction of the external semi-circular canal (more than half of the exposed portion) was found; the dura was opened through the floor of the middle cerebral fossa, attended by the escape from within the dura of a considerable amount of fluid pus, and two iodoform gauze drains were introduced, one as far upward as possible, the other inward along the floor of the fossa.

Shortly after the operation the right pupil became widely dilated, and remained so for two or three weeks, during which time, however, the eye-ground remained normal.

There was a facial paralysis which developed on the second day after the operation, and disappeared to a great extent within a few months. As soon as he was well enough to leave his bed there was found a great unsteadiness of gait, which persisted for two or three months, but finally equilibrium was completely re-established.

On May 28th, a blood culture was taken with a negative result.

On May 26th, there were 18,400 leucocytes, with 75% of polymorphonuclears.

On May 27th, there were 12,600 leucocytes, with 74% of polymorphonuclears.

On May 29th, there were 10,000 leucocytes, with 76% of polymorphonuclears.

On May 31st, there were 10,500 leucocytes, with 70% of polymorphonuclears.

The temperature a few hours after the operation reached 105°, the following day 103°, and the same eight or nine days later, the rest of the time it varied from 101.5° down to normal, at which latter point it remained after June 9th or 10th, two weeks after the operation.

In this case the pathway of infection was unquestionably through the external semi-circular canal and internal ear to the intra-dural space. In spite of the fact that the blood examination gave evidence of a fair amount of resistance on the part of the patient, yet the symptoms preceding operation, together with the escape of so much intra-dural pus at the time of operation, combined to make the case a seemingly hopeless one.

I saw Mr. W. about 10 days ago, April 22nd, 1910, and found his facial paralysis almost cleared up, equilibrium apparently perfect, and what was of particular interest to me, that he hears as well, or better, than before his operation.

The tuning forks from the vertex were heard entirely in the operated ear, and the absolute duration of the C fork (128 v. s.) by air conduction 0, by bone conduction 20″; the C^{III} fork (1024 v. s.) by air conduction 8″, by bone conduction 14″. Showing, I would say, normal labyrinthine hearing, and that his cochlea had not been involved at the time of his acute labyrinth infection.

CASE II. Mr. D., aged 62, was sent in to my service at the Long Island College Hospital, January 11th, 1910, from Setauket, Long Island, where he had been following his occupation as an oyster planter until five days previously. On that day, and increasingly since then, he was found to be distinctly aphasic, and complained of pain in the left ear and side of head. There was scanty discharge from both ears, and Dr. McCrae, who was in charge of him at his home, believing that the drainage from the left ear was insufficient, made a free incision of the left tympanic membrane three days before he came to the hospital.

I first saw him late in the evening of January 11th, and found him so deaf and so aphasic that it was virtually impossible to obtain any information from him. His brother informed me that he had had recurrent ear trouble in his right ear for many years, and in the left ear for not more than four or five months. He had not been specially ailing during the winter, but had attended to his very exposing work until five days ago.

There was pus in both external canals, an old perforation in the right membrane, and the recent, still open, incision in the left. No evidence of mastoid tenderness could be elicited. Temperature was 101°, pulse 76, respiration 20. Frequent hot douching of the ears was ordered, also a Calomel purge.

The next day, January 12th, he slept at intervals during the day, without much evidence of pain, although he could not speak so as to be understood. Temperature 8 a. m., 101°, at 12, noon, 104°, at 8 p. m., 102.4°. Pulse about 100 through the day.

He was seen about noon at my request by Dr. Browning, who leaned toward a diagnosis of cerebral abscess rather than meningitis, but advised waiting a day or two for more definite data as to localization. For January 13th, the bedside record is "that he slept at intervals, temperature lower, unable to keep patient in bed, left ear seems tender to touch. Urine scanty, oz. XXVII in 24 hours." Temperature variation from 102.4° in the morning down to 100.6° at midnight. Pulse 76 to 100.

For January 14th, it is noted that "he slept all night, dozed at intervals through the day, slight reddish discharge from the left ear, takes nourishment better, tongue heavily coated, urine oz. XXII in 24 hours. Temperature at 4 a. m., 100.6°, noon, 99.5°, then rose steadily until at midnight it was 104°, the pulse remaining from 80 to 100. The following morning, January 15th, at 4 a. m., the temperature was 104°, but dropped by noon to 100°.

The eye-grounds were examined on January 12th, and showed a neuro-retinitis of both eyes, slightly more marked in the left, indicating a general increase of intra-cranial pressure. Urine examination was negative. Blood examination showed 18,500 leucocytes, with a polymorphonuclear percentage of 81.5%.

Operation was undertaken January 15th, at 1.30 p. m. The left mastoid was opened, and some granulations and soft bone were curetted from the antrum; the incision was carried upward, and a large area of dura exposed with the

rongeur; upon opening the dura, the pia was found to be intensely congested, and well forward, over the anterior temporal convolutions, was found a large area of exudate held within the meshes of the pia; some of this exudate was removed, and from it there developed the pneumococcus in pure culture. The underlying structures were investigated for any localized collection of pus, but none was found. The patient died about 48 hours later.

In the meantime the patient had been increasingly difficult of restraint, the temperature had gradually risen to 105.5° and the left side of the face became swollen, the tongue became thick, and was drawn (or pushed) toward the right.

About eight hours after death an autopsy was performed by Dr. Murray. A discolored, softened, area of bone was found in the roof of the antrum, which was presumably the path of the infection. The base of the middle lobe on the left side was covered with an exudate similar in appearance to that found at the site of the operation, and another similar large patch covered a considerable portion of the parietal lobes on the right side. There was no abscess anywhere, and the exudate spoken of was nowhere of such a consistency as to be characterized as **liquid** pus, nor is it conceivable to my mind that material of this kind is in the slightest degree likely to make its escape through any such opening in the dura as has heretofore been recommended for the relief of this class of cases.

CASE III. Peter H., aged 24, a deck-hand, was admitted to the Long Island College Hospital, January 29th, 1910. There was a history of pain in the right ear three weeks ago, followed after about two days by discharge. He had suffered somewhat from dizziness, but had walked a considerable distance to and from his work until the day before he entered the hospital. On the same day he seems to have developed a facial paralysis.

He was admitted to the hospital late in the evening of January 29th, with a temperature of 105°, was unconscious, the neck was rigid, the limbs also were flexed and rigid.

On January 30th, I saw him about noon, Dr. Browning

about 4 p. m., facial paralysis was marked, neck and limbs rigid, some indistinct evidence of mastoid tenderness, there was pus coming through a seemingly sufficient opening in the tympanic membrane, no sagging of canal wall.

He was entirely irrational and there was involuntary urination. There was a leucocytosis of 25,000, and a polymorphonuclear percentage of 85. A spinal puncture was made, and about 1 oz. of turbid spinal fluid was withdrawn.

Urine examination showed some albumen present, and casts taking on a granular form.

Dr. Browning and I concurred in the diagnosis of meningitis, and we both felt that the evidences of its general fulminating character were so unmistakable as to make an operation at that time an entirely hopeless undertaking.

Temperature at 4 a. m. 102°, 8 a. m. 103.6°, 12 m. 104°, 4 p. m. 105°, 8 p. m. 104°, midnight 103.5°, with a pulse ranging from 82 to 108.

January 31st, still unconscious, very restless and noisy, urination involuntary. Takes no nourishment. Temperature 103.5° at 4 a. m., down to 100.4° at 4 p. m. and up to 102° during the evening, with a pulse varying from 80 to 100.

February 1st, very restless and noisy most of the night, still urinates involuntarily. Spoke a few distinct sentences in the morning, becoming more rational as the day advanced, and began taking nourishment. Temperature at 4 a. m. 101°, and slowly fell to 98.6° by midnight, with a pulse from 80 to 90.

From this time there is nothing very worthy of note except that the patient's condition improved from day to day. For a week he complained of severe pain in the left side of head—he developed a boil in the external canal.

By February 14th he was so far convalescent as to be up and about the ward—temperature and pulse were normal—headache virtually gone. February 12th, a blood examination showed 20,000 leucocytes, and 81% of polymorphonuclears—mastoid symptoms gone—tympanic membrane

healed and hearing returning. There was no growth from the spinal fluid.

This case would seem to me to be somewhat of an upset either to our ideas of diagnosis, or to those of prognosis, of otitic meningitis; either we cannot be certain of a diagnosis of meningitis in the presence of practically all its recognized symptoms, even including that of an opaque spinal fluid, which I have heretofore looked upon as final and conclusive, or else we must modify our ideas of prognosis and admit that an occasional case may recover even without operative interference; and if we choose this latter horn of the dilemma then may the question not be asked with some show of justification, how many of the reported recoveries after operation might have recovered if left alone.

Another question arises in my mind as perhaps worthy of our consideration, as a result of the findings in cases one and two of this series, and of observation in other cases on the operating and autopsy tables; are there not two kinds of otitic suppurative meningitis? One class in which fluid pus is found within the dura and in which drainage offers some hope of cure, and another class in which the pus, or exudate, is held suspended within the meshes of the arachnoid or pia, and in which drainage offers but little, or no, prospect of relief?

Are they two distinct processes, or only different stages of the same process? If different processes, does one present a possibly better prognosis than the other? And if so, can we attach to each a sufficiently distinctive combination of symptoms to enable us to determine beforehand in which cases operation may or may not be wisely undertaken?

A CASE OF TYMPANIC VERTIGO.

By J. E. SHEPPARD, M. D., Brooklyn, N. Y.

Miss M., aged 26, first consulted me September 29th, 1909, upon the recommendation of Dr. Frederick C. Eastmann.

She stated that for two years she had been treated by several specialists in Brooklyn and New York, and that a diagnosis of Ménière's Disease had been repeatedly made.

Previous history. Over two years ago she had a moderate attack of dizziness, and three or four months later began to grow dull of hearing in the left ear, the deafness having since then steadily, though slowly, increased. About six months after the first attack of vertigo she had a second attack, followed by another two or three months later. These attacks have increased in frequency and severity until now she averages four or five attacks daily; often wakes up dizzy during the night; on two occasions has fallen (both times backwards); has never lost consciousness, but usually feels drowsy after, and sometimes before, the attacks; has frequently vomited during the attacks; some time after the trouble began constant tinnitus was added to the other symptoms, but does not seem to have been especially worse during, or after, the attacks of vertigo. Two or three times a month she has rather severe headaches, mostly frontal, and not apparently connected with the dizzy attacks. No evidence of stomach, bowel or uterine trouble. She wore glasses when at school, but gave up their use nearly ten years ago.

Tuning fork tests—from mid-line, vertex and teeth, both C and C^{III} forks were heard better in the unaffected ear. The absolute duration and relative intensity tests, I would say, indicated "mixed," middle and internal ear, disease of only moderate severity, certainly **not** a pure labyrinth lesion. Watch $8''/72''$. Speech and whisper 20 feet. The

tympanic membrane was moderately retracted and opaque, with a rather small light reflex.

On using Siegel's Otoscope, the membrane moved freely, *excepting only* the manubrium mallei, which remained quite stationary, and on rarefying the air in the canal some dizziness was caused. The Eustachian tube was shown by the catheter and diagnostic tube to be perfectly free and dry.

In the nose the following abnormal conditions were found: The septal cartilage, high up, was either split or covered with a greatly thickened mucosa, probably the former, so that there was some pressure between it and the right middle Turbinate. The left middle Turbinate cystic, probably the remains of an old ethmoiditis.

In the naso-pharynx was found a rather thick adenoid pad, scarcely enough, however, to be obstructive to nasal breathing.

I recommended to Dr. Eastmann that, first of all, on account of the frequent headaches and the former use of glasses, the eyes be eliminated as a probable source of the vertigo, and they were accordingly examined by Dr. Edward Wright, who reported astigmatism of both, but more of the left, and ordered glasses, which were worn two or three weeks with seeming relief of the headaches, but with absolutely no effect on the vertigo.

Consequently on October 19th I advised as the next step in our process of elimination, and this recommendation was made solely because of the above-mentioned immobility of the malleus handle as shown by Siegel's Otoscope, that a link be removed from the chain of ossicles, and the incus was at once removed in the office under cocaine anaesthesia.

On November 18th, one month later, I made the following note: "After the first day or two of slight upset following the operation, and as long as the opening remained in the tympanic membrane, the patient was entirely free from vertigo and was able to go out and about with more assurance than at any time during the past year." During the next few days after the membrane healed, and this I am at a loss to explain, she had a few mild attacks of vertigo, and I

was just on the point of recommending, as the next procedure, that the adenoid pad be removed, when the vertigo ceased, and she has had no further attack from that time to the present.

The tinnitus remains unchanged, and there has been a moderate increase of the deafness.

That this case should never have been classed as one of Ménière's Disease, properly so-called, will, I think, be readily admitted by all of us, both from the character of the attacks, if carefully analyzed, and from the tuning-fork tests.

But I am not so sure of my ground when I endeavor to trace the connection between the immobility of the hammer handle and the interference with equilibration: my reasoning was that along with, and probably on account of, this immobility in the outer part of the chain, it was perhaps fair to assume that the foot-plate of the stapes was more or less firmly pressed into its niche in the oval window in such a way as to interfere with the tension of the intra-labyrinthine fluid, through a disturbance of the compensatory provision of the membranes of the round and oval windows spoken of by Dr. Blake in his paper presented last week to the American Laryngological, Rhinological and Otological Society.

Discussion.

Dr. Edward B. Dench, of New York City, thought the cases of meningitis reported by Dr. Sheppard exceedingly interesting. The first case, which was cured by operation, was certainly the exception rather than the rule, and everyone having such a case should record it. One was apt to conclude that these cases are not relieved by operative interference, and only by collating a number of cases could a satisfactory method of procedure be formulated. He could not understand how the infection took place through the labyrinth in this case, and believed that further examination would have proved it to have taken place in the middle cranial fossa. The condition seemed to him to have been

one of localized labyrinthitis, and meningitis, the route of infection of which was through the middle cranial fossa. Cases in which a specific germ is found in the cerebro-spinal fluid are very unfavorable for operation. In CASE III no germs were found. Such cases, even when serious, do sometimes spontaneously recover, as did this one. The symptomatology as ordinarily laid down in text-books is misleading. Meningitis is ordinarily supposed to be associated with a very high temperature,—an impression which should be corrected.

The case of tympanic vertigo was interesting. He had seen a number of these cases. In the only one upon which he had operated he removed the malleus, incus and drum membrane, and he tried to remove the stapes, but it broke. The patient was extremely dizzy for about ten days after the operation, but this cleared up, and he has had only one attack of vertigo since the operation. He is able to ride horseback, to play tennis, and to attend to his business. The speaker had asked Neumann if he ever performed the tympanic operation for the relief of vertigo, and the reply was that he did not, as the only way it would cause relief would be by hemorrhage into the labyrinth, which would give an absolutely dead labyrinth. In the case just cited the labyrinth was not put out of commission. Relief can often be obtained in these cases by operative interference, by middle ear operations. It should be remembered that in these cases of vertigo where there is an anomaly in the conducting mechanism, the labyrinth is rendered more irritable than usual. Slight changes in blood pressure will determine vertigo, which, under normal conditions, would not be the case. Glonoin in small doses will cause immediate cessation of vertigo.

Dr. James F. McKernon, of New York City, recalled a case of purulent meningitis which he had reported before the New York Otological Society about five years ago, and which was published in the *Archives of Otology*. The cerebro-spinal fluid was not only flaky, but it contained very

large numbers of meningococci. These were demonstrated not only by microscopic examination, but by pure culture. The case finally recovered, after six weeks. If the laboratory findings in such cases cannot be accepted, then laboratory methods are certainly faulty.

Dr. Wendell C. Phillips, of New York City, did not agree with Dr. Pierce concerning the recovery of cases of diffuse meningitis. He cited one case of recovery in which there were undoubted evidences of this disease. The case had been elsewhere reported in full.

Dr. S. MacCuen Smith, of Philadelphia, was especially interested in the case of tinnitus aurium mentioned by Dr. Sheppard. The late Dr. Charles H. Burnett was the first to advocate and to practice removal of only the incus to cure tympanic vertigo, he said: The procedure proved very successful. The speaker agreed with Dr. Pierce concerning the recovery of diffuse meningitis.

Dr. Frederick L. Jack, of Boston, cited two cases, one of tympanic vertigo, and one of otitic meningitis. The first was that of a hack driver who had been the rounds of the clinics seeking relief from vertigo which was so severe that he would fall off his hack two or three times a week. He had received all manner of non-surgical treatment, without relief. He was told that the symptoms might be relieved by operation, but that his hearing would probably be lost. Stapedectomy was performed, the patient remained in the hospital two weeks, and for the two months in which he was under observation there was no return of the trouble. In the second case, when he was called in consultation the patient was *in extremis* with symptoms of meningitis, and operation was not advised. The patient recovered. A year later he had acute inflammation in the opposite middle ear, and was sent to the hospital, where he died. Autopsy revealed well marked signs of meningitis on both sides, one healed, the other active.

Dr. Sheppard, in closing the discussion, said that in localized meningitis there is presumed to be a wall shutting off the disease from the general meninges, and this wall is supposed to be not very far away from the opening made into the pus. In the case cited Dr. Stickle made the opening, and when he put the gauze in above and beneath as far as he could make it go and still found pus, the case was considered one of diffuse meningitis. This could not be proved, however. The cerebro-spinal fluid was certainly opaque, and the symptoms were entirely different from those of cerebral abscess. In the vertigo case the reactions were not made.

THE VACUUM CLEANER. ITS USEFULNESS IN ROUTINE OFFICE WORK AND IN THE OPERATING ROOM. DESCRIPTION OF APPARATUS.

W. H. HASKIN, M. D., New York, N. Y.

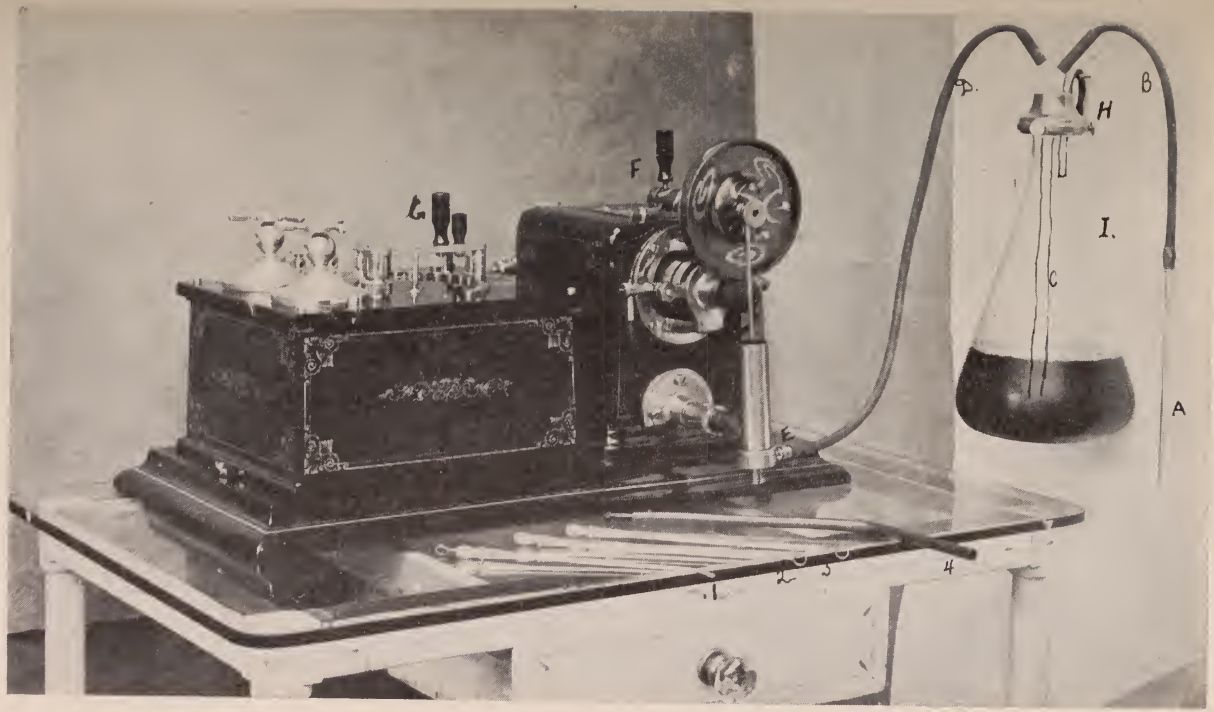
For the past seven or more years I have used suction so constantly in my practice it has never occurred to me that others had not found its great usefulness also, but I find that my confreres, in New York at least, have not made use of it, and so thought it might prove to be interesting, at any rate to those already possessing a pneumo-massage apparatus. The simple demonstration of its ability to suck up a raw egg will prove that it will remove any secretion that may be found in the nose or throat. In my hands it has been found to be of the greatest service in all operative work upon the mastoid and in the nose and throat. I believe that there is a very wide field for its use in general surgery, especially in abdominal work.

Vacuum cleaning is being used very extensively in the household, why not apply it in our own work? There are three pneumo-massage pumps on the market, The Victor Electric Co.'s, The Wappler Co.'s, and one made in Germany which is handled by the Kny, Scherer Co. The only addition necessary is to insert a vacuum bottle between the pump and the site to be cleaned, as per illustration which has been made for me by the Victor Co., who have also devised a very simple bottle and set of five catheters, made for them by the Hardy Co. I have always used the soft silver Eustachian catheters of various sizes.

The insertion of the bottle secures a uniform suction and prevents the materials removed from entering the pump and thus interfering with its action. If any one will use the apparatus a few times and become accustomed to it I feel sure that he will recognize its value and will find many uses for it in daily practice. The treatment of all

suppurative cases is greatly simplified, for the perfect removal of all secretions seems to relieve the congestion caused by them, and the application of whatever medicament desired will prove doubly efficacious as it actually reaches the membrane and is not neutralized by the secretion. Cases of pan-sinusitis are especially suited for its use and I know of nothing that will so thoroughly cleanse cases of atrophic rhinitis, although it may be necessary to soften the thick crusts in the early treatments. It will remove the thick adherent mucous on the pharyngeal walls. The relief afforded in emptying the cavities of peri-tonsillar abscesses is great and aids materially in cutting short the distressing symptoms. In fact, it will clean out any abscess cavity better than any other method I have ever used and cause less distress at the same time. In the ear I cover the end of the catheter with a small piece of capillary rubber tubing which lessens the danger of hurting the patient if he should move suddenly. It is the only way that I have ever found to absolutely remove the muco-purulent secretions from the middle ear and attic. It would take too much time to mention all the conditions where this method of cleaning will be found useful, that is in office work.

Perhaps its greatest field for use will be found in the operating room. I always use it in my mastoid operations, rarely having to use gauze or other sponging material. It keeps the field cleaner and one never has to stop during the operation after the assistant has become accustomed to handling it. When working in the tympanum the operator can hold the catheter in his left hand and keep the field where he is working absolutely clear. In the after treatment of these cases the attic, middle ear and the wound itself can be kept cleaner and with less injury to the delicate granulations than by any other method. It took me some time to find how large a catheter was necessary for its successful use in the tonsil and adenoid operation, but I believe that the large one shown will be found most useful, and it is none too large. It is fine in intra-nasal operations of any kind, and with a suitable catheter ought to be most



A.—Catheter.
B.—Rubber tubing. Connects catheter with long glass tube.
C.—Long glass tube.
D.—Rubber tubing connecting vacuum bottle with the pump.
E.—Movable valve for reversing the pump action.
F.—Switch for starting the pump.
G.—Switch for turning on electric current.
H.—Metallic top with two outlets for glass tubes and a hook for hanging the bottle wherever desired.
I.—Vacuum flask.
1&4.—Metal catheters made by The Hardy Co.
This pump is part of the Victor Company's Pneumo-massage, cautery and light apparatus.

helpful in the radical sinus operations, but I have never had one to do and I attribute this largely to the successful treatment which I have been able to secure with suction. In routine office work it is well to keep the vacuum bottle half filled with some strong antiseptic solution and to have the glass tube, which is connected with the tubing from the catheter, extend nearly to the bottom of the bottle. By this means the tubing can be emptied and sterilized at the same time by merely reversing the pump and thus forcing the solution through it. This enables you to see just what has been removed at the same time. When used in operations, there should be no solution, or at least very little, in the bottle, and the glass tube must be kept well away from the bottom. If this is not done the blood will be quickly stirred into froth and this will get into the pump and cause trouble. I make no claim for originality of any kind in showing this, but I do hope that it will give others as much help and real pleasure in their work as it has given me for several years. I should think that it would extremely useful in direct laryngeal, broncheal and oesophageal work, for the pump could easily be attached to a small tube such as is used at present.

THE VENOUS SYSTEM OF THE LABYRINTH.

By GEORGE E. SHAMBAUGH, M. D., Chicago, Ill.

In my work on the blood supply of the labyrinth of the domestic pig (1) I found that all the veins of the labyrinth drained through one channel, the vein of the aquaeductus cochlea. Previous to this work it had been accepted generally that the veins left the labyrinth by three routes, the vein of the aquaeductus cochlea, the vein of the aquaeductus vestibule, and a third vein in the internal meatus. Siebenmann in reviewing my first work suggested that my failure to find the vein of the aquaeductus vestibule might be due to a rapid method of corrosion I had employed which he thought might have destroyed this vessel. That this was not the case I was able to demonstrate by the examination of preparations in which no corrosion of the capsule was necessary, but where the capsule was still cartilage and could be made transparent in creosote.

Later I worked out the blood supply in the labyrinth of the sheep and the calf. (2) I was unable to find in either of these species a vein in the internal meatus which drained blood from the labyrinth. In the sheep I found that all the veins of the labyrinth drained as in the labyrinth of the pig, through the vein of the aquaeductus cochlea. In the calf, however, I did find occasionally as an anatomical variation, a small vein leaving the labyrinth along the aquaeductus vestibule.

In 1908, K. Asai, (1) working in the laboratory of Prof. Siebenmann, published the results of his investigation into the blood supply of the labyrinth of the dog and the rat.

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(1) Distribution of Blood-Vessels in the Labyrinth of the Ear of Sus Scrofa Domesticus. The Decennial Publications, University of Chicago. The University of Chicago Press, 1903.

(2) The Distribution of Blood-Vessels in the Labyrinth of the Ear of the Sheep and the Calf. Archives of Otology, Vol. XXXIV, 1905, also Zeitschrift f. Ohrenheilkunde Vol. XLVIII, 1904.

(1) Anatomische Hefte, Bd. 36, 1908.

In both of these species he found practically the identical distribution of venous trunks leaving the labyrinth as Siebenmann had previously described for the human ear, (2) that is the existence of three channels; the vein of the aquaeductus cochlea, the vein of the aquaeductus vestibule, and the vein of the internal meatus.

My own interest in the subject led me to undertake to verify these findings in the labyrinth of the dog. I secured two litters, fifteen pups in all, and made injections through the carotid. I succeeded in getting a number of quite perfect injections in which all the veins and capillaries as well as the arteries were filled. In none of these preparations was I able to discover a vein in the internal meatus that drained blood from the labyrinth. I did find in all of my preparations a vein leaving along the aquaeductus vestibule. This vein was not an anatomical variation as I had found in the calf but a constant anatomical finding. This vein of the aquaeductus vestibule did not, as shown, in my best preparations, drain the blood from the vestibule and semi-circular canals as illustrated in the drawings of Asai, receiving all the veins from the semi-circular canals as well as from the maculae of the utricle and saccule. In the two most perfect of my preparations the vestibular veins were exactly alike. The arrangement is shown in this drawing and the preparation under the microscope. Here the vein of the aquaeductus vestibule drains the crus commune and the posterior crus of the horizontal canal. The anterior vestibular vein drained the anterior crus of the superior canal and received a tributary from the anterior crus of the horizontal canal, and at the same time drained the maculae acusticae of the vestibule. The posterior vestibular vein in both of these preparations drains the posterior crus of the posterior canal as well as the anterior crus of the horizontal canal by a vein crossing the vestibule along the anterior surface of the utricle.

For several years I have been making injections of the

(2) Die Blutgefasse in Labyrinthe des Menschlichen Ohres. F. F. Bergman. Wiesbaden, 1894.

human embryos as I have been able to secure material. Among these I have succeeded in making a number of excellent injections of the veins as well as of the arteries. Here also I have not succeeded in finding the vein in the internal meatus draining the labyrinth. The vein of the aquaeductus vestibule was constant, as in the labyrinth of the dog. Its tributaries from the semi-circular canals varied, however, markedly in different labyrinths.

The typical arrangement of the veins draining the semicircular canals appears to be as follows: a vein from both the anterior and the posterior crus of each canal and in addition a separate vein draining the crista acusticae of the ampulla. This vein of the crista did not, in most of my preparations, empty into the vein of the aquaeductus vestibule but joined either the posterior vestibular or the anterior vestibular vein.

TWO CASES OF LABYRINTHINE DISEASE FOLLOWING CHRONIC SUPPURATION (CHOLESTEATOMA).

By NORVAL H. PIERCE, M. D., Chicago, Ill.

Case I. Male, laborer, aged 47. Suppuration in left ear about forty years. Ten days before admission to hospital he had an attack of vertigo, which confined him to bed three days. Vertigo of a diminished degree was still present, but no ataxia apparent. Patient gave history of sores on penis many years ago, which healed without other manifestations.

Examination: Total loss of left tympanic membrane, granulations on promontory, pus from attic; total deafness. Right tympanic membrane depressed, but intact; low limit G-2, high limit 4.5, Edelman-Galton. Weber to left. Nystagmus: Spontaneous horizontal to right; slight rotary to left. Rotation to right gave slight increased rotary nystagmus to left. Rotation to left: marked, mixed to right. Caloric (cold) marked on irrigation of right. Doubtful on irrigation of left. No fistula symptoms on tests. Dec. 8, 1909, radical operation. Patient took the anesthetic (ether) very badly. Respiration arrested several times. Hemorrhage profuse from soft parts and bone delaying operative procedures. Some necrosis below knee of Fallopian canal. No fistula found. Cholesteatoma in rather small antrum. Internal ear not opened.

December 11th: Condition excellent. No spontaneous nystagmus.

December 22nd: After left rotation, rotary nystagmus; to right, 30 sec. Rotation to right, slight nystagmus, to left. Vertigo absent. Patient complains of a slight dull "drawing" sensation in left occiput. General condition excellent. Has been walking around since Dec. 13.

Feb. 16, 1910: Spontaneous rotatory nystagmus, left. No spontaneous nystagmus, right, but normal reaction after rotation to left. Rotation in both directions causes vertigo.

Rotation to right with head forward, 90°, increases spontaneous nystagmus to left.

Feb. 17th: Vertigo. Vomiting. Slight delirium. Temperature 100°. Pulse 64. Eyes turn to right.

Feb. 18th: Spontaneous horizontal nystagmus to right, practically continuous. Spontaneous nystagmus, rotary, to left; longer strokes than right, but not so lasting. Patient standing or sitting falls to left regardless of position of head. Reflexes approximately normal, but left hand awkward in finger to nose test. Mental sluggishness. Slight delirium and slight thickness of speech. Diaphoresis. Bilateral choked disks. Nystagmus not affected by hot or cold irrigation of left ear. Pulse ranged between 65 and 75. Indefinite tenderness over left occiput.

Feb. 20th: Radical labyrinth operation (Neumann's). Chloroform narcosis. Labyrinth full of granulation tissue, flakes of cholesteatoma and pus. Dura of posterior fossa smooth and of normal color. No fluid escaped on incision. Thorough exploration of cerebellum in all directions for from 3 to 4 c. m. negative. No facial paralysis. Patient died in twelve hours without regaining consciousness, and in convulsions.

Urine occasionally contained albumin, but no sugar or casts. Temperature after first operation, 100° F., dropping to normal on the following day, and remaining so until Dec. 21st, when it arose to 99.2°. Thereafter subfebrile, and pulse averaging 90 until Dec. 27th, 101.4°, pulse averaging 100. February 17th, temperature 100°, pulse 67. Leucocytes 15,000.

Post-mortem was held on the morning of February 21, 1910. Brain removed and placed in formalin solution. No meningitis. Normal sinuses. Brain, with cerebellum, was cut after some weeks by Dr. Le Count, who reports: In the white substance of the left cerebellar hemisphere a cavity is found containing a fluid which is probably pus, measuring three centimeters antero-posterior and two and one-half centimeters from side to side. Inner wall reaches the median line. The lateral wall is three centimeters from the surface,

while the dorsal wall is only one-half centimeter below the cortex. The wall of the cavity is of grayish-white color and extends into the cerebellar substance to a depth of from one to two millimeters. Outside of this discolored zone is a narrow hyperemic zone, easily distinguished. The cavity is on its inner surface occupied by detritus which it is impossible to identify without microscopical examination. Evidently an old abscess produced by a micro-organism, which set up very little inflammatory reaction in the surrounding tissue. It is probably of hematogenous origin, there being no macroscopic connection between the labyrinthine inflammation and the abscess cavity.

The interesting points in the case are, first, the apparent reaction of the semi-circular canals on the left side to the caloric test. While this was not marked, it was still present and it would lead one to think that possibly a remnant of function may be present in the static apparatus, even when it is largely destroyed. The patient at no time complained of marked headache, but notwithstanding the fact he must have had the abscess at the time he entered the hospital. Indeed, it is a question whether the abscess did not begin from a thrombotic cortical vessel at the time he had his severe vertigo, ten days before his admission to the hospital. While it was known that the left labyrinth was involved at the time he was admitted, cerebellar abscess was not suspected.

It is curious to note the disappearances of the labyrinthine symptoms and even the improved condition of the patient after the first radical operation. The recurrence of nystagmus and vertigo followed the day on which the statis apparati were so thoroughly tested, and but for the pathological findings, which proved the abscess to be a chronic one, it was in my mind that possibly the shaking-up of the diseased static apparatus caused a spread of the inflammation, which led to his death. The case above all accentuates the importance of carefully testing the static apparatus in all cases of chronic suppurative otitis, in order to distinguish in the

after-course of the patient between involvement of the labyrinth and the cerebellum.

The question arises as to why cerebellar abscess was not evacuated at the time of operation. Surely it was not because of lack of thoroughness in exploration. While at the present time I am unable to positively say that the exploring knife entered the cavity, yet I am convinced that this must have happened and that either the fluid making up the contents of the cavity was so thick that it would not flow, or that the peculiarities of its walls closing in immediately after the knife was withdrawn prevented its escape, very much as the corn pulp in our battleships prevents the ingress of water after a perforation of the hull.

Another question which arises is, Was the abscess due to the labyrinthine suppuration or was it hematogenous? At the present time we can positively say that it did not arise directly by extension from the labyrinth, as healthy cerebellar substance to the extent of three centimeters intervened between the surface in contact with the labyrinth and the abscess wall. Dr. Thor Rothstein is of the opinion that it is highly probable that the infection came from thrombosis of the blood vessels descending from the cortex in the upper surface of the cerebellum.

Macroscopic examination of the left labyrinth shows that it was full of granulation tissue and pus and cholesteatomatous tissue. This is now hardening and a microscopic examination will be made. There was no fistula extending from the posterior surface to the posterior fossa, nor was there apparently any inflammatory reaction in the internal auditory meatus. The invasion of the labyrinth occurred probably through one or the other fenestrae but this point awaits microscopic confirmation.

The right labyrinth, macroscopically, was normal.

Case II. Admitted January 12, 1910. Male, farmer, aged 21. Discharge from left ear for fifteen years. Left ear totally deaf; right ear, whisper eighteen feet. Low limit, G-2; high limit, 1. Weber to right. Schwabach +

10 seconds. Rinne, + 15. Left canal constricted. Purulent stinking discharge. Granulation tissue. No ataxia. No history of vertigo. Spontaneous nystagmus to right. Caloric tests: Left negative; right normal. Rotation to right, very slight nystagmus to left. Rotation to left, nystagmus to right, 30 seconds. No fistula symptoms.

Jan. 15th: Radical mostoid. Cholesteatoma. Uneventful course.

Feb. 16, 1910: Vertigo on rotation in both directions. Some spontaneous rotary nystagmus in both directions. Rotary nystagmus to left increased after turning to right. Rotary nystagmus to right increased after turning to left.

March 5th: Anode, 7 ma., to left mastoid; nystagmus right.

Cathode, 12 ma., to left mastoid, nystagmus left.

Anode, 7 ma., to right mastoid, nystagmus left.

Cathode, 12 ma., to right mastoid, nystagmus right.

Nystagmus to left after rotation to right, 20 seconds.

Nystagmus to right after rotation to left, 25 seconds.

Discharged, March 5th, with dry cavity; slight moisture from Eustachian tube. Gain in weight.

This is a case in which a serous (toxic) labyrinthitis existed as a consequence of chronic cholesteatoma of the left ear. It is interesting to note the reactions of the labyrinth before and after operation. In this case there was no history of vertigo, which strongly contrasts with the foregoing case. The spontaneous nystagmus to the right may be explained on the theory that the functional activity of the left static apparatus was depressed. The caloric tests of the left would tend to prove this; also the rotation test. This is contrary to the dictum that a serous labyrinthitis is always irritative, in which case the static apparatus is more easily stimulated than normally.

After the operation the left static function regained its normal reaction.

It is interesting, as exemplifying a case of labyrinthine involvement in which an operation on the labyrinth is contra-indicated.

Discussion.

Dr. Edward B. Dench, of New York city, mentioned a case which had come under his observation at the New York Eye and Ear Infirmary, in which there was some vertigo and also some spontaneous nystagmus to the sound side. The elaborate tests mentioned by Dr. Pierce, were not made because the patient's condition rendered immediate operation imperative on account of acute symptoms. There was a history of a chronic middle ear suppuration of a long duration. When the patient was placed upon the operating table, a small swelling was found just posterior to the lobe of the affected ear. This was at first supposed to be a sebaceous cyst, and an effort was made to extirpate it prior to making the ordinary incision for the radical operation. On incising the integument, the tumor was found to contain foul-smelling cheesy material. The characteristic cyst-wall of a sebaceous tumor was wanting. The radical operation was then performed. and the entire mastoid was found to be filled with a cholesteatomatous deposit. This cholesteatomatous mass had perforated the inferior wall of the external auditory meatus, and from this perforation in the floor of the canal, a fistula extended downward to the tip of the lobule. The supposed sebaceous cyst was simply a continuation of the cholesteatomatous deposit in the middle ear and antrum. On performing the radical operation, it was found that the cholesteatoma had perforated the horizontal semi-circular canal and vestibule, and had also destroyed that portion of the promontory lying between the oval and round windows. In other words, a complete labyrinthine operation had been performed by Nature. Recovery, in this case, was uneventful.

Dr. Dench said that the question of nystagmus to the sound or affected side, depended entirely upon the char-

acter of the lesion. If the labyrinth were completely destroyed, then there would be nystagmus to the healthy side. On the other hand, if the labyrinthine lesion were recent, nystagmus would occur toward the diseased side.

Dr. Arthur B. Duel, of New York City, failed to see how Dr. Dench could have suspected that a spontaneous nystagmus would have been found, had he looked for it, in a chronic case of the type described. The only time when this may be suspected is in the acute stage. These chronic cases of suppuration in the labyrinth never give rise to spontaneous nystagmus.

Dr. Geo. E. Shambaugh, Chicago, said that involvements of the labyrinth occuring in suppurative middle ear disease are of special interest to all of us just now because of the advances that have been made in the diagnosis of such labyrinth involvements, and in the surgery of the labyrinth. It is necessary that we keep clearly in mind the fact that while there are a number of pathological conditions occurring in both the acute and chronic suppuration of the ear which give rise to symptoms of greater or less severity, indicating an extension to the labyrinth, there are but few of these cases that call for an operation on the labyrinth itself. In our care of cases of suppurative ears presenting symptoms of labyrinth involvement, it is important first of all that we diagnose the type of labyrinth involvement from which the patient is suffering. In the second place it is important that we keep clearly in mind those conditions which justify an operation on the labyrinth; i. e., a case of suppurative otitis media, either acute or chronic, may develop an acute serous labyrinthitis with very pronounced labyrinth symptoms: such a case, however, should not have the labyrinth operated on. In another case there may be an erosion producing a fistula into the labyrinth, and where there is present what we designate as a circumscribed labyrinthitis running along for many years with here and there an acute exacerbation with very marked labyrinth symp-

toms. An operation on the labyrinth in these cases is also unjustified. The only condition that with our present knowledge warrants our opening the labyrinth is the condition arising from a diffuse suppuration of this structure. The differential diagnosis between the various conditions which involve the internal ear in both the acute and chronic suppurations of the middle ear is not always easy, and it is in these cases where the scientific training of the otologist in the physiology of this complicated mechanism is of great assistance. The diagnosis of a diffuse suppuration of the labyrinth is not, however, so difficult since this apparently always means a complete destruction of function both in the cochlea as regards hearing and in the vestibular apparatus as regards its function of equilibrium. A practical diagnosis of total deafness can as a rule be readily made, and with the caloric tests the diagnosis of a complete destruction of function in the vestibular apparatus can also be made.

THE ACCESSORY CAVITIES OF THE NOSE IN THE FIVE YEAR OLD CHILD WITH NOTES ON CASES OF SUPPURATION OF THE ANTRUM OF HIGHMORE AT AND BEFORE THAT AGE.

By WILLIAM C. BRAISLIN, M. D., Brooklyn, N. Y.

An attempt is made in this paper to give the degree of development of the accessory nasal sinuses in the five year old child with the comparative development of the same regions as they exist at birth, together with notes on suppuration of the antrum of Highmore in cases at, or before five years of age.

At birth the accessory nasal sinuses present as follows:

Maxillary antrum (of Highmore) is present as a cavity practically filled with its own lining membrane.

Ethmoidal spaces, consisting of one small anterior cell on either side (the ethmoidal bullae) lined with membrane, and connected with the nasal canal by an opening, another anterior ethmoidal cell and a small posterior cell on either side.

Sphenoidal spaces absent.

Frontal spaces absent.

THE ACCESSORY CAVITIES OF THE NOSE OF THE NEW BORN.

The maxillary antrum exists at birth as a space in the upper jaw. The space, however, is practically filled by reason of the reduplication of the lining membrane. It may be entered, from the nose, on dissection, with a small probe. On slight dessication, from exposure to the air, the lining membrane shrinks sufficiently to demonstrate a considerable

bony space. The maxillary antrum in the new-born thus revealed is in close apposition to the inner lower wall of the orbit, having, when stripped of its lining membrane, an anterior-posterior diameter of ⅜ of an inch and of the same or slightly less diameter from its inner lowest point upward and outward to its highest. In one section photographed, (Fig. 3), the saw was carried through the natural opening which leads downward and outward from the middle

Fig. 1. Vertical section through body of spenoid of newborn. P.P. posterior nares. 1 inch from front free edge of alveolar border.

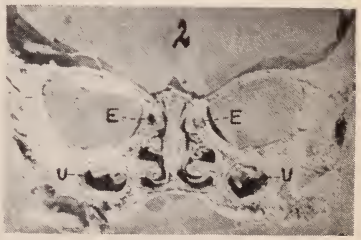


Fig. 2. Vertical section through posterior ethmoidal cells E.E. U.U. unerupted molars. ¾ inch from front free edge of alveolar border.

Fig. 3. Vertical section through maxillary antra A.A., ostium O.; uncinate process U.; unerupted deciduous bicuspids T.T.; ½ inch from front free edge of alveolar border ethmoidal bullae E.E.

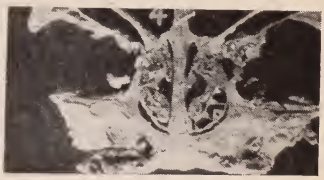


Fig. 4. Vertical section through nose of Newborn, ¼ inch behind free alveolar border. D. dilatation of lower end of lachrymal duct. The breath space lies between middle and lower turbinates.

meatus into the antrum. This opening, which in the new-born is a sinus of some length, passes above the uncinate process.

The floor of the antrum is relatively higher, as compared with the floor of the nasal canal, than in the adult. The region of the superior maxilla, anteriorily, inferiorily and posteriorily to the maxillary antrum, is taken up by the

unerupted teeth, deciduous and permanent. Thus, at birth, the teeth fill a great part of the available space within the body of the superior maxilla. The teeth are soft and pulpy, much more so than the individual bony spaces in which they are enclosed, but show a beginning calcification of the enamel, particularly at their inferior borders.

The development of the accessory nasal cavities may be regarded as taking place as a sequence to the rapid development of the superior maxillary bone—an urgent necessity for the proper accommodation of the development of the teeth.

In the lower forms of animal life, the upper jaws are more fully developed at birth than is the case in the human species. In the latter, by reason of the larger cranial development, the face suffers, **ante partum,** a consequent retardation; but after birth there follows a correspondingly rapid development of the bones of the face. Thus in the three months' old infant the maxillary antrum is found the size of a small hazel nut.

Subsequently to birth, the development of the accessory nasal spaces, consequent to the development of the bones of the face, takes place in the following order:

Maxillary antrum (antrum of Highmore), present at birth.

Ethmoid cells, four present at birth.

Sphenoidal cells, develop later than the fifth year.

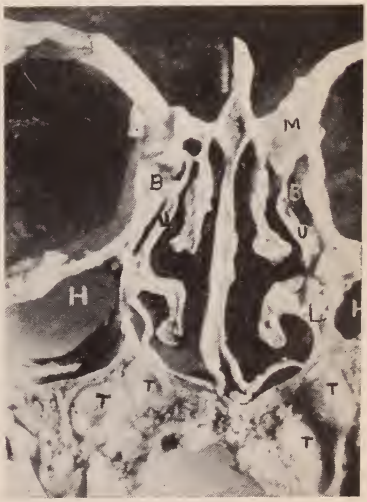
Frontal sinuses develop subsequently to fifth year (about age of puberty.)

Normally, at the end of the third year of life, all of the ten upper and ten lower deciduous or milk teeth have been erupted.

From the third to the sixth year occurs a period of quiescence of tooth eruption, though not of the growth of the teeth; about the end of the sixth year the eruption of the permanent teeth begins, of which the first to appear are the (first) molars, followed in order by the central incisors (7th year), lateral incisors (8th year), first bicudpids (10th

year), second bicuspids (11th year), canines (12th or 13th year), second molars (12th to 15th year).

From the end of the third to the end of the sixth year no teeth erupt. Closely crowded within the body of the bone, external to, and below, the antrum and the nasal floor, the teeth steadily increase in size during this period, and the size of the superior maxillary bone which encloses them receives the stimulus of growth, in like ratio.



Figs. 5 and 6. Section through anterior part middle and inferior turbinates (½ inch behind front incisors) of five year old child.

From behind: M.M. membrane covering infundibuli (opening at I.I.) B. ethmoid bulla. U.U. front pt. uncinate process. L. lachrymal duct. W.W. front walls of maxillary antra. T.T. unerupted (permanent) teeth, or sockets of same.

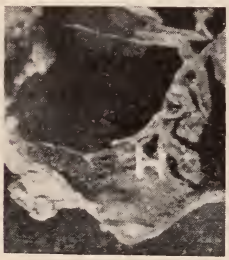
From front: M. membrane covering anterior ethmoidal cells. U.U. front pt. uncinate processes, forming vestibules to the maxillary ostia. H.H. maxillary antra. L. lachrymal duct. T.T. unerupted (permanent) teeth, or their sockets.

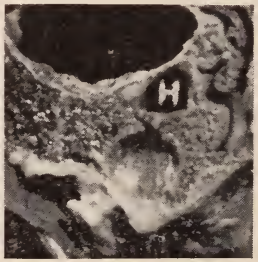
MAXILLARY ANTRA IN FIVE YEAR OLD CHILD.

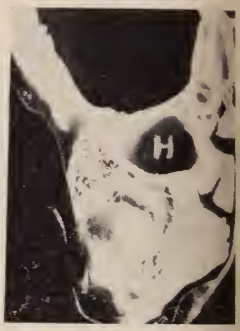
At the fifth year of age the maxillary antra have reached a considerable size. Following the outline of the orbit, they have extended upward and downward so that at the lowest they are nearly on a plane with the floor of the nose. They have extended anteriorily and posteriorily at the fifth year of life, measuring in this, their greatest diameter, ¾ inch or slightly more.

The relationships of the maxillary antrum may be well delimited by the orbit and by the erupted teeth. At five years of age the maxillary antrum extends forward to the plane of the anterior edge of the canine tooth; posteriorily to the posterior edge of the (unerupted) first permanent molar. Its superior wall follows closely the outline of the inner lower wall of the orbit. Most of its floor is still at a higher plane than the floor of the nose.

Cross section of the cavity gives a roughly drawn right-angled triangle, the right angle being at the intersection of







Figs. 9 to 12. H. Maxillary antrum in close relation to wall of orbit. Photo from behind.
7 mo. embryo. At birth. 1 yr. old. 5 yrs. old.

the lower and inner walls of the antral cavity. Thus the inner wall is nearly vertical, the inferior, roughly horizantal, the supero-external wall follows the outline of the orbit.

ETHMOIDAL CELLS OF THE FIVE YEAR OLD.

In the child of five years and over, well developed ethmoidal cells, both anterior and posterior, are found. The infundibulum, a modified ethmoidal cell, is found closely beneath the most depressed part of the bridge of the nose, extending upward and outward for a short distance beneath the floor of the anterior cerebral fossa, just external to the cribriform plate. Posteriorly to the infundibulum lies the bulla ethmoidalis, at this age a cell of considerable size. It

is worthy of remark that the uncinate process, forming a sort of gutter for the ostium of the maxillary antrum, is well developed at this age. Several ethmoidal cells exist on either side posteriorly to the ethmoidal bulla, the last but one, of large size, lying beneath the attachment of the middle turbinal. Between this cell and the anterior sphenoidal surface of the corresponding side lies the posterior ethmoidal cell.

Figs. 7 and 8. Section at posterior nares (1¾ to 2 inches behind front incisors) of five year old child.

From behind: Body of sphenoid of diploëtic bone, but lacking membranous lined spaces. Arches of posterior nares low, more triangular than in the adult.

From front: Body of sphenoid composed of diploëtic bone. B.B. sphenoid.

SPHENOIDAL CELLS ABSENT IN THE FIVE YEAR OLD.

The greater part of the body of the spenoid is as yet a mass of diploëtic bone, and, as yet, shows no space worthy of particular mention except the indenture of the posterior ethmoid cell.

It is curious to observe that the frontal sinus, as yet unformed, must develop by a rather different process from that of the sphenoidal cells. In the latter, their formation apparently proceeds by means of the absorption of the diploëtic bone tissue, within the body of the sphenoid. While in the case of the frontal sinus the bone tissue at this age

remains so thin that it allows no spaces to form within the inner and outer tables. The infundibulum plainly presents its upper extremity in the direction of the future frontal sinus, so that it appears that the frontal sinus is formed by an outward and upward extension of the infundibulum as the inner and outer tables of the frontal separate.

CASES OF SUPPURATION OF THE MAXILLARY ANTRUM IN EARLY CHILDHOOD.

The cases of suppuration of the maxillary antrum in early childhood, here referred to, are briefly brought to notice because in one, the case of a five year old child, an autopsy was done; in another, a very young infant, a wide destruction of tissue took place, with recovery, but with some resultant deformity of the face.

In the child of five years the case was first seen post-mortem, and no satisfactory history, previous to that time, could be obtained. None of the relatives spoke English, and nothing satisfactory was learned of the case during the few hours he passed in the hospital. Extensive necrosis of both superior maxillae was found with extensive destruction of the soft tissues of the mouth and palate. The anterior portion of the triangular cartilage of the septum was the seat of destructive inflammation. The right maxillary antrum contained dark, bloody, purulent fluid, as did also the right nasal canal. There was supposed to have been a tubercular meningitis accompanying this condition, but such was found not to be the case.

Another case demonstrates the mischief which an infection of the maxillary antrum in a young babe may produce.

The case was a girl baby of three months of age. The first symptoms noticed by the attending physician referable to the nose were, a swelling, and eversion of the lower eyelid and a purulent nasal discharge for about two weeks following an attack of bronchitis. The cheek had been punctured with a bistouary below the right lid and a small amount of pus had been evacuated. The eversion of the lid still continued and the purulent nasal discharge had persisted. At

this time I first saw the child. The antrum was entered through the nose and pus was evacuated. From this time treatment was continued by the attending physician, who several times kindly reported to me the progress of the case. He stated that pus continued to discharge from both the cheek and the nose, and later from the alveolar process for some weeks, with intermittent improvement, and that a sequestrum of the bony shell of the alveolar process was removed. Recovery is now complete.

A case of maxillary-ethmoidal abscess in a four year old child, seen since the above was written, presented a profuse purulent discharge from the eye on the side corresponding to the purulent nasal discharge, which persisted for a time subsequent to operation and after entire cessation of the nasal discharge.

SUMMARY.

Development of the maxillary antrum proceeds coincidently with the development of the superior maxilla and other bones of the face, the size of which, independently of the child's age, may be superficially inferred from the appearance of the external features.

The unerupted teeth within the superior maxillary bone occupy space which prevents a full development of the maxillary sinuses to their full size up to adult life; or, in other words, until after the eruption of the third molars, or wisdom teeth. At an early age, even immediately after birth, the maxillary antrum is capable of becoming infected and thus the seat of abscess.

The close relationship of the eruption of the teeth to the development of the maxillary sinus is worth bearing in mind, since the more teeth already erupted, the larger the antrum of Highmore will be found. In operations on the maxillary antrum in early childhood, the nasal route would seem the most natural and accessible one, since the antrum, though small, is readily located just exteriorily to the attachment of the lower turbinate between this point and the inner

lower wall of the orbit. Practically, however, due to the intra-nasal space being so encroached upon by the crowded turbinates, I believe the preferable route is that through the canine fossa as close to the wall of the orbit as possible. In opening into the maxillary antrum through the canine fossa, because of the close relationship of the unerupted teeth and its small size, its relationship may be well located, especially the supra-external wall, by its close proximity to the wall of the orbit.

Edematous conditions of the eyelids, with or without purulent discharge from the eye, purulent discharge from the nose, or a history of recent purulent nasal discharge, should be considered with reference to the possibility of abscess of the antrum of Highmore.

REPORT OF A CASE OF TUBERCULOUS MENINGITIS FOLLOWING PURULENT OTITIS MEDIA, AND COMPLICATED BY ANTERIOR POLIOMYELITIS AND MEASLES.

By WENDELL C. PHILLIPS, M. D., New York, N. Y.

B. K., the subject of this sketch, age 22 months, of Russian parentage, had suffered from purulent otitis media in the right ear for three and one-half months at the time she was brought to the New York Post Graduate Hospital on October 11th, 1909.

The mother stated that about eight weeks previously the child had suffered from an acute fever, on account of which she had been admitted to Zion Hospital, where she remained for two weeks. Upon leaving the hospital, it was noted that she was unable to walk or stand. There had been no history of tenderness or swelling about the mastoid process until four days previous to her appearance at my clinic, when a swelling appeared in front and above the right ear. The discharge had a fetid odor and had become more profuse. She also complained of pain in the head, and had been sleepless.

She was admitted to the babies' ward of the hospital, where a general examination was made. Her weight was 24 pounds, temperature 100°, pulse 120 and respirations 36; throat culture negative; urinary examination revealed faint trace of albumen, occasional pus and excess of indican.

Blood examination showed; red cells 4,400,000; leucocytes 15,000, polynuclears 65%. The pus was thin, yellowish and profuse. There were several scabs on the face, chin and right leg, and two scars from abscesses in the left gluteal region. There was marked bulging of the left abdominal wall.

Dr. H. D. Chapin, who made the physical examination, obtained a clear history of anterior polio-myelitis. A simple mastoid operation was performed. There was extensive necrosis and much foul pus and detritus. The inner table covering the knee of the lateral sinus was necrosed, and carefully removed.

During the month subsequent to the operation there was no apparent improvement in the anterior polio-myelitis, the paralysis did not improve, the mastoid wound remained foul, and there was a constant discharge of pus from the external auditory canal. On November 15th she developed measles and was sent to the Kings' County Hospital. She was returned to the babies' ward of the Post Graduate Hospital on the 21st day of December.

Upon re-admission her temperature was 98°, pulse 132 and respirations 28. Her mastoid wound had not healed and the granulations were flabby; there was a profuse discharge of foul pus both from the mastoid wound and the external auditory canal. She now had a right side facial paralysis, her skin was yellow, she had almost constant cough and she was semi-comatose. Each afternoon there was a moderate rise in temperature but never over 101°. At that time it was the writer's opinion that the child was a victim of tuberculosis which had extended to the meninges.

On November 24th a radical mastoid operation was performed. Since the previous operation the necrosis had extended in all directions, but especially had the antrum and attic tegmen become soft. Upon removing the necrosed internal cranial table from the middle cerebral fossa it was observed that the dura was of a dull red color, and covered with small whitish specks which appeared to be miliary tubercles, which were considered confirmatory of the diagnosis of tubercular meningitis.

The round window was found open and pus was seen to exude through the opening. At this stage of the operation the child's condition became so alarming that I was obliged to finish the excavating without delay. This was done and three or four small incisions were made in the dura for the

purpose of drainage. A lumbar puncture was now made, both for the purpose of relieving the intracranial pressure and to obtain a laboratory report of the fluid. The fluid came away under pressure but was clear.

The laboratory report upon a smear of secretion from the wound showed rare intracellular didplococci. A culture from the same secretion gave streptococci and a few staphylococci. The report upon the examination of the cerebro-spinal fluid showed tubercular bacilli, but no other organisms, pus or blood.

On the following day the child was some brighter and more inclined to take nourishment. The temperature reached 102° F. From this time, however, she gradually failed and died from exhaustion on December 31st. The temperature did not rise above 102° at any time subsequent to the second operation.

The points of interest are: (1) the characteristic appearance of the dura, showing miliary tubercles; (2) confirmation of the tubercular condition by the finding of tubercle baccili in the cerebro-spinal fluid; (3) it was the writer's first case of purulent otitis media complicated by anterior polio-myelitis; (4) the combination of purulent otitis media, tubercular meningitis, anterior polio-myelitis and measles is a rare occurrence; (5) at no time was there a high temperature except at the beginning of the attack of measles, when it reached 104°.

THE PRESENT STATUS OF THE ADENOID OPERATION IN CHILDREN

E. TERRY SMITH, M. A., M. D., Hartford, Conn.

For a number of years it has seemed to me that many times operations upon tonsils and adenoids should be performed separately; that too often we perform the double operation without considering the condition of the patient. Much greater immediate benefit is noticed after the removal of enlarged adenoids than after enucleation of the tonsils, so that I firmly believe it is often wiser to first remove the adenoids; then, when the physical condition of the patient is sufficiently improved, the tonsils may be enucleated, if necessary. Too many times children are nearly exsanguinated by the combined operation and for some time are physically worse, showing no effects of improved breathing until months afterward.

Believing it to be the tendency among physicians to more carefully consider cases of this kind, I sent the following circular letter (to which I received two hundred and seventeen replies) to a number of men, both in this country and abroad:

"It has seemed to me that it might be of interest to determine the method of procedure which is adopted by the majority of physicians in operating upon children for adenoids.

We know that some prefer to operate in the office without an anaesthetic and without insisting that the patient be kept quiet afterwards, while others use ether and advocate several days of rest. The manner of operating also seems to vary.

Believing that some general information in regard to this matter would be valuable, I am enclosing a list of questions to be answered and returned at your convenience."

The questions asked were as follows:

"1. Do you use an anaesthetic in operating for adenoids in children?

2. What anaesthetic do you prefer?

3. Do you prefer profound or light anaesthesia?

4. In what position do you prefer to have the patient during the operation?

5. Any suggestions about technique of operation?

6. What instruments do you use?

7. What after treatment, including any general instructions that you may give the patient with regard to breathing exercises, etc.?

8. What is your belief about recurrence?"

From the replies to the **first question, "Do you use an anaesthetic in operating for adenoids in children?"** I gathered the following statistics:

One hundred and seventy-two physicians always use an anaesthetic.
Thirteen use an anaesthetic generally, depending on circumstances.
Six use no anaesthetic usually.
Four use cocaine locally.
Three do not use an anaesthetic.
Three use it sometimes, but not generally, and according to wishes of parents.
Three use it sometimes and sometimes do not.
Two use it, but not invariably.
Two, when patient is unmanageable.
One uses it if the patient is an infant.
One, if the patient is under eight years of age.
One, for those over five years.
One, for those under ten.
One, always for young children.
One, only when necessary.
One, in exceptional cases only.
One, in private practice, not in clinic.
One, not as a rule, but for very nervous children.

The answers to the **second question, "What anaesthetic do you prefer?"** show that **one hundred and thirty-two** phyicians prefer ether, **twenty** chloroform, and **nineteen** gas. The choice of other men is as follows:

Ten prefer a combination of gas and ether.
Eight, ethyl chloride.
Three, ethyl bromide.
Three, somniform.
Two, sometimes chloroform, but usually ether.

Two, sometimes ether, but usually chloroform.
Two, somniform or chloroform.
Two, sometimes one anaesthetic and sometimes another.
Two give first choice to ether, second to chloroform, third to ethyl chloride, fourth to gas.
One prefers ether usually.
One, somniform if tonsils are easily accessible.
One, gas for adenoids and gas-ether if tonsils are included.
One, ethyl chloride, with ether for second choice.
One, chloroform or ether.
One, gas-ether if patient is not too young.
One, ethyl chloride and ether.
One, gas or ether.
One, A. C. E. mixture.
One considers it almost criminal to use chloroform.
One gives ether rarely and ethyl chloride and gas rarely.
One prefers chloroform for beginning, and then ether.
One, chloroform for babies; gas, when over five years.
One, gas, and cocaine locally.

The **third question, "Do you prefer profound or light anaesthesia?"** brought the following information:

Light anaesthesia is preferred by **one hundred and sixty-four** physicians.
Profound preferred by **thirty-nine.**
Medium, by **five.**
Fairly deep, **four.**
Primary, **two.**
Moderately complete, **two.**

I also quote from various letters the following answers to this question:

"Depends on case, often profound."
"Until jaws relax."
"Not too light, fearing danger of reflex."
"Not deep enough to abolish laryngeal reflex."
"Just sufficient to do work well."
"So that reflexes are not quite abolished."
"Enough for complete relaxation."
"Surgical degree."
"Full relaxation."
"Depends on conditions."
"Sufficient for unconsciousness, but not to destroy throat reflex."
"Profound required only in exceptional cases."
"Rather light, not too deep."

"Enough to get muscular relaxation."
"No child should be fully anaesthetized."
"Do not abolish cough reflex."
"Sufficient to insure complete removal."

Fourth question—"In what position do you prefer to have the patient during the operation?"

Lying on back with head lower than body is the position advocated by **eighty-nine.**
Lying on side with head lower than body, advocated by **fifty-four.**
Sitting up, **forty-two.**
Lying on back with head level with body, **twelve.**
On side for adenoids; on back for tonsils, **one.**
On side with anaesthetic; on back without, **one.**
Sitting up if tonsils are submerged; on side in other cases, **one.**
On side with ether; sitting up with gas, **one.**
On side well over to side, **one.**
Left side, **one.**
Horizontal, on back or right side, **one.**
On side with anaesthetic; sitting up without, **one.**
Head higher than body, **one.**
On back, changing to side at times, **one.**
Sometimes on back, sometimes on side, **one.**
Head brought forward to knees when operating, **one.**

Suggestions in regard to technique of operation (fifth question) are quoted as follows:

"Careful exploration with finger to be certain all adenoid tissue is removed."
"Clean out thoroughly." (Dr. Ray, Louisville, Ky.)
"Should always be followed by finger examination and curettage."
"Avoid haste; avoid trauma, and be sure naso-pharynx is clean, finger being necessary to ascertain this."
"Assistant holds patient on knees, with head on chest and hands over head, being first passed under patient's arms."
(Dr. G. D. Murray, Scranton, Pa.)
"Avoid scraping."
(Dr. J. A. Patterson, Colorado Springs, Colo.)
"Strong electric head lamp."
(Dr. F. S. Stillman, Columbus, Ohio.)
"About three sweeps with curette, then massage with finger."
(Dr. Wade Thrasher, Cincinnati, Ohio.)
"Dr. Kyle's nasal stretcher at close of operation, putting one blade in each nostril and separating turbinal walls a little."
(Dr. D. T. Vail, Cincinnati, Ohio.)

"Remove mass with forceps, then the remaining fragments with curette; afterward rub down the whole vault with index finger, taking care to break up all adhesions."

(Dr. A. J. Smith, Yonkers, N. Y.)

"Always follow instrument by finger."

(Dr. J. K. Sterrett, Pittsburg, Pa.)

"Locally, cocaine and adrenalin,—sometimes by injection with curved needle." (Dr. H. Gradle, Chicago, Ill.)

"Barnhill technique, simplest."

(Dr. W. J. Atkinson, Paterson, N. J.)

"Forceps first—curette next." (Dr. R. J. Held, New York, N. Y.)

"Adnoids can generally be removed in mass with the sweep of the curette, if properly used"

(Dr. R. Lewis, Jr., New York, N. Y.)

"Fossa of Rosenmüller must not be forgotten."

"Always a thorough preliminary clearing of Rosenmüller's fossa with index finger, and then use the broadest curette that naso-pharynx will accommodate."

(Dr. J. W. Jervey Greenville, S. C.)

"Have the patient held firmly by two assistants."

"Complete removal, care being taken to clean Rosenmüller's fossa."

"Tonsils first when present."

(Dr. George C. Stout, Philadelphia, Pa.)

"Avoid hemorrhage by having mucous membrane and periosteum intact." (Dr. J. A. Kenefick, New York, N. Y.)

"Thorough excision." (Dr. W. F. Dudley, Brooklyn, N. Y.)

"Do not necessarily confine myself to curette."

(Dr. H. E. Smyth, Bridgeport, Conn.)

"Attend at the same time to any nasal obstruction."

(Dr. H. L. Wagner, San Francisco, Cal.)

"Examine with finger to see if everything is removed; a curette with a good sharp curve." (Dr. Emil Amberg, Detroit, Mich.)

"Invariably digital exploration after treatment."

"Forceps, curette, then gauze sponge on finger to scrub vault."

"Apply mouth gag before giving anaesthetic; be ready to use curette as soon as relaxation begins. Curette quickly and thoroughly and then throw head forward over basin and clear blood from throat."

(Dr. Gordon King, New Orleans, La.)

"Gag in mouth, head promptly bent forward after systematic curetting which should clear vault, same being ascertained by the finger and by forcibly blowing through the nostrils."

(Dr. A. W. De Roaldes, New Orleans, La.)

"I draw soft palate and uvula well up out of the field so that I can see the tissue I am removing."
(Dr. Fred Whiting, New York, N. Y.)

"Gottsten curette, with finger nail for fossa of Rosenmüller."
(Dr. G. A. Leland, Boston, Mass.)

"With or without forceps; then curette and finger."
(Dr. Harold Walker, Boston, Mass.)

"Mouth kept open with mouth gag and tongue held down with tongue depressor; digital examination before terminating operation." (Dr. Cohen, Philadelphia, Pa.)

"Palpation with finger to determine complete removal an important step. Soft palate retracted to inspect wound after operation." (Dr. S. Rosenheim, Baltimore, Md.)

"I dilate nostrils with Kyle-Septon forceps."
(Dr. T. W. Moore, Huntington, W. Va.)

"Be sure to get all." (Dr. J. L. Kent, Lynchburg, Va.)

"Routine—forceps, curette, finger."
(Dr. J. E. Sheppard, Brooklyn, N. Y.)

"Take your time to be thorough."
(Dr. John R. Winslow, Baltimore, Md.)

"Follow curette with gauze."
(Dr. Thomas J. Harris, New York, N Y.)

"No suggestions, except that adenoids should be completely removed."

"I use forceps, then curette, and finally gauze on index finger."
(Dr. John M. Foster, Denver, Colo.)

"Tonsils first, adenoids last." (Dr. D. J. Gibb Wishart, Toronto.)

"Sense of touch constantly used as guide. Finish with rubbing surface with gauze." (Dr. W. A. Wells, Washington, D. C.)

"Always safer to operate at patient's home or hospital. No immediate after treatment in the way of syringing or douching, unless symptoms arise. I never operate in the presence of acute inflammatory process. To avoid secondary hemorrhage would try the calcium lactate given internally forty-eight hours before operating, and continue it for same period afterwards. Good results have been claimed by those using this remedy."
(Dr. Lederman, New York, N. Y.)

"First forceps, then curette."
Dr. Emilio Martinez, Havana, Cuba)

"Central sweep with curette, then curetting with special wiry curette."

"After removal of central mass it is of utmost importance to remove any side masses, curetting with index finger of the left hand as guide." (Dr. H. N. Spencer, St. Louis, Mo.)

"Know that blade of curette is above mass; one clean sweep."
(Dr. Sherman Voorhees, Elmira, N. Y.)

"No suggestions, except that I try to do adenoids first, which makes the resulting bleeding from the more dangerous tonsil vessels less." (Dr. Swain, New Haven, Conn.)

"Care of jaw, teeth, and soft parts."
(Dr. E. R. Holt, Portland, Me.)

"Try to remove in one mass."
(Dr. John Van Duyn, Syracuse, N. Y.)

"By using electric light see what you are doing."
(Dr. J. A. Stucky, Lexington, Ky.)

"Occasionally, finger massage following use of instrument when Rosenmüller fossa is involved."
(Dr. E. W. Fleming, Los Angeles, Cal.)

"Have trained anaesthetist to handle gas. To keep field clear of blood imperative."

"Much bad work is done in removing adenoids. To my mind the curettage, commonly thought right, is a mistake, and for the same reason forceps are bad instruments. They remove the growth, it is true, but they leave a ragged, torn base with irregularities and fragments on which infection is very much more likely to occur, with consequent danger to the ears, and which also favor recurrence of the lymphoid masses. The curette of suitable size and strength for the case in hand, properly used, makes not a curettage or scraping, but a clean, sweeping excision with one stroke, leaving a clean, smooth cut base, surgically correct, and permits the removal of the adenoid mass 'in toto.' The side pockets should not be injured by instrumental force, which must always be ineffective here. They can best be cleaned out by the finger nail. This manoeuvre, where skill has been attained, surpasses all the usual plans for removal of adenoids." (Dr. Charles Graef, New York, N. Y.)

"I make my technique as simple as possible. Under local anaesthesia I require and must have two good assistants who know every step of the work to be done. One of these assistants holds the child in the lap, facing me, and held so firmly that no movement can occur. The second assistant holds the head as if in a vise. With every instrument ready, the entire work is often done in from five to ten seconds, even when both tonsils are removed with a guillotine."
(Dr. John F. Barnhill, Indianapolis, Ind.)

"The only suggestion I have to make in regard to the technique of the operation is the use of a curette, one which is broad enough to fit easily into the nasal pharynx, with a short arm leading from the handle to the blade of the instrument.

The curette should be very sharp and the ring of the instrument should fit firmly around the adenoid. The curette should then be drawn downward with one firm stroke so as to completely sever the adenoid from the pharyngeal wall. In the majority of cases it is not necessary to make more than one cut. If the adenoid is not completely severed and hangs on the pharynx, I remove it with small punch forceps. I am just about convinced that the curette is the only instrument to be used and that the Beckman, with its modifications, is the best. I cannot think that the forceps are of very much practical value in the removal of adenoid tissue."

(Dr. John J. Kyle, Indianapolis, Ind.)

"If the fossae of Rosenmüller are not entirely cleared out, the operation may as well not have been done as far as oral conditions are concerned. This point seems to be frequently overlooked by general practitioners who attempt to do the operation, and by some of the throat specialists who do it for the sake of the breathing spaces alone. I have been preaching this fossa of Rosenmüller complication for twenty years. A certain spasm of enthusiasm for this seems to have taken hold of some members of the profession recently." (Dr. G. A. Leland, Boston, Mass.)

"I believe it is essential that the surgeon take his time and carefully remove all the adenoid tissue without cutting the periosteum. When the periosteum is wounded, I believe that we are very apt to have cicatrization, which leads to drying and discomfort in the nasal passage. For this reason I generally avoid the sharper curettes."

(Dr. George B. Wood, Philadelphia, Pa.)

"Instruments should not be too sharp, neither should they be dull. If too sharp, the mucous membrane is too likely to strip up. If dull, the adenoid cannot be thoroughly removed."

(Dr. D. A. Kuyk, Richmond, Va.)

"The region around the Eustachian tubes should always be cleared. For this purpose a very small, narrow curette of the Beckman type should be used. The best one sold in America is made by the Globe Optical Company of Boston, and was designed by a Boston specialist whose name I do not at this moment recall." Dr. George L. Richards, Fall River, Mass.)

"I operate as rapidly as is consistent with thoroughness; indeed, I would rather do two quick operations than one prolonged one, as I regard excessive or extensive traumatism from sponging, scraping, or other manipulations as a distinct element of danger and source of complication, tending to excessive reaction and inviting secondary infection."

(Dr. W. J. Rogers, Columbus, Ohio.)

"I believe too much is made of thoroughness by some, too little by others. I have frequently, with a little cocaine or eucaine, rubbed with the pulp of the index finger most of the sago-grain collections out through the covering mucosa (as the Knapp roller-forceps squeeze out the trachoma granules) with minimum blood loss and risk. Some of the cases later require formal operation because nasal breathing has not been maintained, perhaps never gained. The children have at times volunteered to kiss me good bye, so the operation cannot be so brutal as some assert. Many are permanently relieved. I always prefer to reduce congested conditions by astringents, especially ten per cent silver. Many obstructions are merely congested conditions and may not be found on the operating table, although on the contrary the enlargement may prove greater than expected, perhaps mainly from effect of the anaesthetic. Needless, sometimes dangerous bleeding may occur from neglect of this preliminary astringing. As the adenoid tissue occupies almost all parts of the vault, often including the upper surface of the soft palate, any one instrument cannot be expected to remove more than the central mass, which is not necessarily the worst part. The scar from its removal draws the lateral masses, if left, into the center for 'recurrence.' Imperfect nasal breathing usually antedates the hypertrophy and like the high arch palate is oftener cause than effect, if indeed palatal deformation is ever the result of adenoids." (Dr. B. Alex. Randall, Philadelphia, Pa.)

"I have observed so many cases of dry rhino-pharynx operated upon in different parts of the country, that I think a note of warning should be sounded to prevent this unfavorable result." (Dr. Thomas F. Gallagher, Denver, Colo.)

The replies to **question six, "What instruments do you use?"** show that **one hundred and twenty-six men** favor both curette and forceps; **seventy-four** use curette only; **seven,** forceps only; **three,** forceps rarely; **one** uses forceps only when necessary to finish; **one,** forceps occasionally; and **one** favors the Schütz adenotome.

In answer to **question seven,** almost all speak of the importance of rest in bed after the operation. Some advise the use of a spray or a cleansing solution of some kind. Almost all agree on a light diet for several days, and many mention the importance of breathing exercises. Some of the many answers to this question were:

"Tonic treatment."
"Instructions according to case."
"After one month if mouth breathing habit continues, use adhesive plaster over mouth."
"Lie on side for two hours so bleeding can be detected."
"Keep mouth closed when walking, and sleep out of doors."
"Attention to occlusion of jaws."
"After treatment symptomatic."
"Have child breathe with mouth closed."
"Let nature alone and always operate in hospital."
"Avoid hemorrhage by two hours of rest."
"Care in blowing nose."
"Use adhesive plaster over mouth if necessary."
"Simple lavage, boric acid solution."
"Thirty per cent argyrol in fossa on P. N. application as soon as bleeding ceases."
"Sleep on side or abdomen first day or two. Suck ice pellets if desired."
"Daily application of argyrol in fossa for several days; usually five grains iodide of iron for several weeks."
"Encourage nasal breathing often by offers of reward."
"No instructions necessary if removal is complete."
"Immediately after operation lie on chest, hips elevated; gargle daily; one or two applications iodo-glycerine after several days."
"Do not blow nose hard. Insist on nasal breathing."
"After treatment depends upon condition of nostrils."
"Spray of antipyrine and Dobell's solution. Plenty of fresh air."
"Advise nose breathing; use normal salt solution wash."
"Nothing after adenoids alone. Simple antiseptic gargle after tonsils."
"Pure air, sunshine and good food, exercise and iron."
"Laxative, night of operation."
"If necessary, have teeth straightened."
"Never operate at office."
"Record temperature for two or three days."
"Laxative, if necessary, and instruct patient to breathe through nose."
"Normal saline solution after second day."
"Dobell's spray and Aristol powder, mouth must be kept shut, tonic frequently."
"Use bland oil in nose for six months; make application to nose five or six times."
"Instruct how to blow nose, and breathe, and, if needed, use adhesive plaster to keep mouth shut."
"Keep mouth closed as much as possible."

"Mild alkaline wash; general tonic; fresh air and sunshine."

"Always nose breathing, and treatment of nose, if necessary, to remove obstructions."

"Simple after treatment of anaesthesia, and breathing through the nose; sometimes menthol albolene spray."

"Usually treat catarrhal condition that is always present."

"Wear bandage or use adhesive plaster to establish nasal respiration."

"Chin bandage or strap on the lips at night."

"Hot alkaline gargle, instructions to keep nose well evacuated and mouth closed."

"Caution in regard to habit of mouth breathing after obstruction from adenoids has been removed."

"Quiet two days for adenoids, longer for tonsillotomy."

"Plenty of outdoor exercise."

"Breathe through nose after a few days as much as possible."

"No medical treatment unless specially indicated. Deep breathing with closed mouth."

"Cold bathing and open window."

"Keep mouth shut."

"Never use nasal sprays in after treatment."

"No immediate after treatment; instruct not to blow nose."

"Mentholated drops for nose and throat spray. Re-education for nasal breathing."

"After three days an antiseptic wash and argyrol may be applied; after this, cultivate nasal breathing and secure same by adhesive plaster at night."

"Keep mouth closed at night with plaster."

"Try and form the nose breathing habit."

"Use iodoform or Aristol ointment and give zinc oxide to be sniffed up nose. If nasal respiration is not good, teach child how to use nose."

"Mouth closed and, if necessary, apparatus worn at night."

"If mouth breathing persists after the operation and no other obvious cause such as deflected septum, engorged turbinates or deformed maxilla can be determined, I instruct the parents to remind the child frequently of the necessity for keeping the mouth closed. In the course of time this results in correcting the defect." (Dr. Robert Levy, Denver, Colo.)

"My after treatment consists of rest in bed for a couple of days. Then I prefer my patients to remain either in a clean room or out in the open air, where there is no dust, but I desire that they shall be considered convalescent for at least a week before they are permitted to enjoy family parties, social gatherings, car rides, school, or anything where they may come in contact with possible infection such as diphtheria,

scarlet fever, etc. In fact, isolation within reasonable degree, as well as rest combined with pure air, constitutes my only after treatment, save that of looking after the condition of the stomach and bowels. I regard any inefficient attention to hygienic conditions after this operation as well-nigh criminal."

(Dr. Lefferts A. McClelland, New York, N. Y.)

"An alkaline wash is given through the post-nasal space, if possible, but always through the nostrils. A plentiful supply of fresh air day and night. Deep breathing exercises. Syrup iodide of iron is most helpful in all these cases."

(Dr. D. A. Kuyk, Richmond, Va.)

"I invariably lay out a course of home treatment by the nurse or mother, laying much stress on overcoming the mouth breathing habit, and assuring the parents that the child will not breathe through the mouth or snore at night once it has learned and acquired the habit of nasal breathing during its waking hours. I give an oil spray to be used two or three times a day, or oftener if necessary, and forbid nasal douches and aqueous sprays. Cold baths or spongings over the chest, breathing gymnastics, setting-up drill, swimming motions, and so on, with tightly closed mouth, warm, light clothing, and plenty of outdoor exercise even in 'bad' weather are also insisted upon. If this regimen is carried through, adenoids will rarely 'come back.' But I warn the parents that there may be recurrence if the mouth breathing habit is not broken up and the child's nose kept clean and free. Recurrences are not seen often since we have been using the curette and getting the growths out 'en masse.'" (Dr. Percy Fridenberg, New York, N. Y.)

"Many children have to be taught nose breathing. I have found a strip of adhesive plaster over the mouth at night to be a useful adjunct. I have also used the device of Dr. French of Brooklyn, made by A. E. Riker, 1204 Broadway, New York. I never operate in my office, but always at the home or hospital." (Dr. George L. Richards, Fall River, Mass.)

"Prohibit any after treatment except mild carbolic acid gargle in case the tonsils have been removed. I believe that any efforts to apply treatment to nose and post-nasal space are uncalled for and may assist in carrying infection to the middle ear. Warn the patient not to blow the nose with force for several weeks after the operation."

(Dr. George E. Chambaugh, Chicago, Ill.)

"I believe that the after treatment is almost, if not quite, as important as the operative measures themselves. It is my habit, if possible, to establish the use of a cleansing spray

for a brief period before the operation. These cases are then entered in one of the hospitals where I have a service, the day before the operation, and as carefully prepared for an anaesthetic as in the case of a major operation. The operation is done the following morning on an absolutely empty stomach, even to the extent of excluding water. It is followed by complete rest in bed on liquid nourishment for twenty-four hours, and longer if the hemorrhage has been unusual. Bits of ice are kept in the mouth perhaps one-fourth or one-third of the time during the remainder of the day, and by afternoon, if bleeding has stopped, a hot antiseptic gargle is employed every hour until the patient is asleep. If there is no reaction the patient is allowed to be up the following day, and generally returns home on the second or third day. The use of the sprays is resumed when there has been no bleeding for twenty-four hours and is employed three times a day for two or three weeks. The hot gargle is continued for two or three days at intervals of one or two hours when the patient is awake, after all exudate or evidence of reaction has subsided. As most of these cases must be regarded in the light of local manifestations of a constitutional condition, they are usually placed on some form of tonic treatment, extending over a period varying from two or three months to two or three years, and an effort is made to classify the atomizer with the tooth brush as a toilet measure and to secure its more or less regular use." (Dr. W. K. Rogers, Columbus, Ohio.)

"I usually give very little after treatment. I instruct the patient to remain quiet, to keep the bowels free, and to eat light food for a few days. I sometimes order a few drops of twenty-five per cent solution of argyrol dropped through each nostril twice a day for a few days."

(Dr. John F. Barnhill, Indianapolis, Ind.)

"The child should be instructed to blow the nose and this should be done frequently during the first few days after operation. Breathing exercises are of much importance in cases where the development of the chest has been impaired."

(Dr. Walter Dowie, Glasgow, Scotland.)

From the answers to **question eight, "What is your belief about recurrence?"** I find that those who do not believe in recurrence, if adenoids are thoroughly removed, number **thirty-two**; those who believe in recurrence, but qualify their statements, **one hundred and fifty-five**; those who

believe in recurrence in children under five years, even after thorough operation, **twenty-nine.**

Some of the replies to this question were as follows:

"Rare in thorough operation." (Dr. Blake, Boston, Mass.)
"Small percentage." (Dr. Wagner, San Francisco, Cal.)
"Occasionally." (Dr. Hale, Nashville, Tenn.)
"Seldom in cases with non-adenoid tendency." (Dr. Page, New York, N. Y.)
"Seldom, if well done." (Dr. Walter Park, Harrisburg, Pa.)
"Due to mouth breathing." (Dr. Borden, Hartford, Conn.)
"Two to three per cent." (Dr. Dudley, Brooklyn, N. Y.)
"Very infrequent." (Dr. Johnson, Paterson, N. J.)
"Rare, if properly performed." (Dr. Emerson.)
"Two to three per cent." (Dr. Arrowsmith, Brooklyn, N. Y.)
"Less frequent in older children ." (Dr. Worthington, Baltimore, Md.)
"Diseased, submerged faucial tonsils, if not entirely removed, will often cause recurrence." (Dr. Chappell, New York, N. Y.)
"If teeth are irregular so as to cause mouth breathing, orthodontia often necessary." (Dr. Chenery, Boston, Mass.)
"Adenoids occasionally recur, especially in lymphatic cases." (Dr. Jervey, Greenville, S. C.)
"One-half of one per cent." (Dr. Ingals, Chicago, Ill.)
"Occasionally." (Dr. Loeb, St. Louis, Mo.)
"Occasionally." (Dr. Milligan, Pittsburg, Pa.)
"Seldom recur if properly removed." (Dr. Robert Lewis, Jr., New York, N. Y.)
"Occasionally." (Dr. Kennon, Norfolk, Va.)
"Especially in lateral fornices if not thoroughly removed." (Dr. Held, New York, N. Y.)
"Rare." (Dr. Atkinson, Paterson, N. J.)
"Less than five per cent." (Dr. A. J. Smith, Yonkers, N. Y.)
"About ten per cent, due to taking cold before parts are healed." (Dr. Vail, Cincinnati, Ohio.)
"About one-half of one per cent." (Dr. Thrasher, Cincinnati, Ohio.)
"Not common." (Dr. Thompson.)
"Better to recur than to use forceps." (Dr. Murray, Scranton, Pa.)
"Sometimes, where constant dyscrasia exists." (Dr. Scott Renner, Buffalo, N. Y.)
"If nasal obstruction is present." (Dr. Fetterolf, Philadelphia, Pa.)

"Seldom recurs after thorough operation." (Dr. Webster.)
"Eight to ten per cent." (Dr. Winslow, Baltimore, Md.)
"When not properly removed." (Dr. Sheppard, Brooklyn, N. Y.)
"Seldom after second year." (Dr. Craige, Montreal, Canada.)
"A certain amount recurs." (Dr. Sherman.)
"Fat children and strumous subjects." (Dr. Cott, Buffalo, N. Y.)
"Often when not thoroughly removed."
(Dr. J. L. Kent, Lynchburg, Va.)
"About two per cent."
"Almost nil when thoroughly done."
(Dr. Chamberlain, Cleveland, Ohio.)
"Rarely, when thoroughly removed."
(Dr. Moore, Huntington, W. Va.)
"Unusual, if well removed." (Dr. Rosenheim, Baltimore, Md.)
"Rare, if operation is complete." (Dr. Reik, Baltimore, Md.)
"In several years, when there is constitutional dyscrasia."
(Dr. Ross, Montreal, Canada.)
"Dependent on lymphoid tendency, providing they have been thoroughly removed." (Dr. Oppenheimer, New York, N. Y.)
"Often." (Dr. Collins, Brooklyn, N. Y.)
"In those given to lymphoid conditions."
(Dr. Harmon Smith, New York, N. Y.)
"About two per cent." (Dr. Porter Jones, Newport News, Va.)
"Very seldom." (Drs. McReynolds and Teay, Dallas, Texas.)
"Possible, but not frequent." (Dr. Taylor, Wilkes Barre, Pa.)
"Occasionally." (Dr. Dorland Smith, Bridgeport, Conn.)
"If imperfectly removed." (Dr. Walker, Boston, Mass.)
"Three and one-half to four per cent, cause mouth breathing."
(Dr. Leland, Boston, Mass.)
"About ten per cent." (Dr. May, New York, N. Y.)
"Some do." (Dr. Lovell, Worcester, Mass.)
"Very slim, if well done." (Dr. De Roaldes, New Orleans, La.)
"Usually do not recur after good operation."
(Dr. Morse, Boston, Mass.)
"Almost never, if thoroughly done."
(Dr. Throwbridge, Fresno, Cal.)
"Very seldom, if properly done." (Dr. Shaw, Boston, Mass.)
"Imperfect after treatment accountable."
(Dr. McAuliff, New York, N. Y.)
"Rare, when completely removed."
(Dr. Butler, Philadelphia, Pa.)
"About two per cent." (Dr. McKennie, Washington, D. C.)
"Possible, but not probable." (Dr. Blodgett, New York, N. Y.)
"Occasionally." (Dr. Timberman.)
"Occasionally." (Dr. Bryan, Washington, D. C.)
"Very unusual." (Dr. Knapp, New York, N. Y.)

"Most unlikely, if radically removed."
(Dr. Spencer, St. Louis, Mo.)
"Never when operation is thorough."
(Dr. Gallagher, Denver, Colo.)
"Sometimes." (Dr. Harlan, Baltimore, Md.)
"Rare." (Dr. Wendell Phillips, New York, N. Y.)
"One in four hundred." (Dr. Berens, New York, N. Y.)
"One-sixth of one per cent." (Dr. Freudenthal, New York, N. Y.)
"One in fifty." (Dr. Chambers, Jersey City, N. J.)
"Exceptional but not infrequent." (Dr. Ring, New Haven, Conn.)
"Small number of cases." (Dr. Crouch, Baltimore, Md.)
"In lymphatic types." (Dr. Voorhees, Elmira, N. Y.)
"If subject to lymphatic diathesis."
(Dr. Dudley, Los Angeles, Cal.)
"Rare." (Dr. Allen, Portland, Me.)
"May, if faucial tonsils are not thoroughly removed."
(Dr. Brown, Columbus, Ohio.)
"Likely to recur in children with lymphatic diathesis."
(Dr. Ellis, Los Angeles, Cal.)
"Rare, if done properly." (Dr. Joachin, New Orleans, La.)
"Very seldom." (Dr. Wells, Washington, D. C.)
"Usually operator's fault." (Dr. D. C. Gibb Wishart, Toronto.)
"Rare, when thoroughly removed." (Dr. H. S. Hedges.)
"Rare." (Dr. Foster, Denver, Colo.)
"Ten per cent in three thousand cases."
(Dr. Theisen, Albany, N. Y.)
"It rarely happens." (Dr. Haskin, New York, N. Y.)
"Very few cases." (Dr. Griffin, New York, N. Y.)
"Rare, when operation is complete."
(Dr. Harris, New York, N. Y.)
"As a rule no recurrence since using the curette."
(Dr. Luc, Paris, France.)
"As to recurrences, they do not take place when the operation is properly performed. There may be instances when circumstances prevent a completely satisfactory operation, and under such circumstances, of course, recurrences are more likely to happen."
(Dr. Lefferts A. McClelland, New York N. Y.)
"I think that recurrence happens only when the operation has been imperfectly performed. At least, I attribute it to my imperfect technique when I have such conditions to deal with." (Dr. John J. Kyle, Indianapolis, Ind.)
"I believe that absolute removal of adenoid tissues in every case is impossible; that after thorough removal with forceps or curette or both, the vault should be thoroughly scrubbed with the index finger covered with coarse sterile gauze.

When this technique is thoroughly carried out, recurrences, where not unheard of, will be exceedingly rare in children operated upon as late as the sixth or seventh year, while if we are forced to operate earlier, recurrences will be less rare." (Dr. Wm. B. Mason, Washington, D. C.)

"Recurrences do take place and they are not always the result of incomplete technique. The regeneration of the adenoids depends upon the continued activity of the identical factors inducing increased functional capacity and increased production of lymphacytes; in other words, this local increase in lymphacytes is due to a continuation of those local conditions which originally brought into play an increased lymphacytic supply and produced the pathologic increase in the adenoid tissue. The causes underlying this phenomenon vary in different cases and the nose will usually furnish factors which have to do with the production of adenoids, so-called, and which, unless corrected, will tend toward a recurrence." (Dr. S. J. Kopetzky, New York, N. Y.)

"Recurrence in adenoids is always in some other man's cases, as malaria and mosquitoes, in New Jersey, are always in the next town." (Dr. Percy Fridenberg, New York, N. Y.)

"I am doubtful as to the question of recurrence. I do not deny that it may happen, but in my own experience I am quite sure that in cases where I have operated for recurrence I did not remove all of the growth the first time. Unless the curette is very sharp, it is likely to leave portions behind." (Dr. George L. Richards, Fall River, Mass.)

"As to recurrence, my own feeling at present is that remnants of adenoid tissue may hypertrophy, and in a very few cases the hyperplasia seemed to amount to as much as the original structure." (Dr. George B. Wood, Philadelphia, Pa.)

"In most cases, so-called recurrence is due to incomplete removal of the hypertrophied adenoid tissue at time of operation. The strumous tendency will favor the growth of any adenoid tissue left." (Dr. Walter Dowie, Glasgow, Scotland.)

"Recurrence undoubtedly in small per cent, oftener in clinic patients under poor surroundings. Always some lymphoid tissue left after operation which may become hypertrophied. Deformed teeth, jaws, palate, etc. predispose to recurrence. If jaws are deformed, advise their correction. Breathing exercises, regularly, advocated." (Dr. Willis S. Anderson, Detroit, Mich.)

"I believe the likelihood of recurrence is slight, if the adenoid is thoroughly removed with a curette. Recurrence is undoubtedly frequent when the adenoid is lacerated and partly bitten away as is often done with forceps. I never use the

term 'adenoids.' There is but one adenoid in any individual. If this is properly surrounded and removed by a well selected curette, by one sweep of the curette, the likelihood of recurrence is not great."

(Dr. John F. Barnhill, Indianapolis, Ind.)

"Recurrence in children with enlarged glands may be expected, especially in very young children. If instructions were followed for a reasonable length of time, recurrence would not be frequent, but care is relaxed as soon as improvement begins." (Dr. D. A. Kuyk, Richmond, Va.)

"Recurrences do take place, but very rarely. I believe that this point in your paper might be enlarged upon. It is not so uncommon to hear of a patient, whose tonsils and adenoids have been successfully removed and in whom the peculiar physiognomy associated frequently with an exaggerated enlargement of these structures exists, being subsequently operated upon by some one else for the same condition. My attention has recently been called to a case of this sort where the patient has been subjected to two subsequent operations and is being prepared for a third one now. I think this phase of the subject should be emphasized, so that general practitioners will understand that the mere presence of an adenoid face or the persistence to some extent of the habit of mouth breathing in a case that has been operated upon is not necessarily any evidence that there is a recurrence, or, what amounts to the same thing, that the operation has not been successfully performed. The point I am making is that cases are often subjected to unnecessary subsequent operation because of the persistence of some of the characteristic symptoms of the post-nasal obstructions." (Dr. George E. Shambaugh, Chicago, Ill.)

"In regard to the question of recurrence, I am inclined to believe that it will seldom happen after a clean operation, and yet I am sure that it sometimes occurs, particularly in children of marked lymphatic diathesis who, notwithstanding the most careful medical and surgical attention, continue to be subject to recurrent colds or similar infectious conditions. Should it take place, it should be dealt with just as in an original manifestation, remaining under more careful scrutiny afterwards. It would seem to require no argument or discussion to establish the fact that a well equipped operating room is the safest place for the performance of any surgical operation, especially one in which severe hemorrhage or sudden respiratory obstruction or cardiac inhibition may be expected in a certain number of cases. I have never seen a physician's office or a private residence properly equipped to deal

with these emergencies, which involve not only appliances that are seldom at hand but also the necessity for absolute rest afterward, under very profound opium narcosis necessitating the skilled observation of a trained nurse and an intelligent interne. I venture to say that few operators get through a hundred cases without being glad to have this sort of hospital environment at hand for at least two or three of them." (Dr. W. K. Rogers, Columbus, Ohio.)

"Negative, if nares are free."

(Dr. A. P. Voislawsky, New York, N. Y.)

"Not one-half of one per cent when work is thoroughly done. This work, however, must take into consideration the presence of nasal obstruction. When both fossae are even partially occluded, recurrence of adenoids is not only more likely, but the results of the operation are unsatisfactory to patient, parents, and physician."

(Dr. F. E. Hopkins, Springfield, Mass.)

"If the child is the subject of lymphadenia, adenoids are apt to recur, no matter how carefully they are removed, though the more carefully the operation is done, the less the likelihood of recurrence." (Dr. W. H. Dudley, Los Angeles, Cal.)

"Recurrence takes place in my experience in a small percentage of cases. In children who are operated upon under four years of age, and in some cases where constitutional dyscrasia exists." (Dr. W. Scott Renner, Buffalo, N. Y.)

"Never recur if removal is complete."

(Dr. Wm. L. Ballenger, Chicago, Ill.)

"If nasal breathing is insisted on and persistently maintained, return is rare. If child is under two years, or if teeth are irregular so that nasal breathing is hard and mouth breathing is the rule, then they will recur more or less. Orthodontia is often essential to restore nasal breathing."

(Dr. W. E. Chenery, Boston, Mass.)

"They do recur in a few cases."

(Dr. W. F. Chappell, New York, N. Y.)

"Exceedingly rare." (Dr. E. M. Holmes, Boston, Mass.)

"They certainly do recur." (Dr. J. A. Kenefick, New York, N. Y.)

"I find them recurring frequently in very young children."

(Dr. Fred Whiting, New York, N. Y.)

"Uncommon in my experience."

(Dr. George T. Ross, Montreal, Canada.)

"Rare, if operation is complete." (Dr. H. O. Reik, Baltimore, Md.)

"Do not believe they recur."

(Dr. Lewis A. Coffin, New York, N. Y.)

"My belief about nearly all recurrences is that they are due to incomplete removal. Our recent routine (the past five years) is to do practically all our adenoid operations in the office, in the upright position, under ethyl chloride, and to allow the patient to go home in about an hour.

(Dr. J. E. Sheppard, Brooklyn, N. Y.)

"Rare, when complete operation has been done."

(Dr. Thomas J. Harris, New York, N. Y.)

"Seldom, if ever, after radical removal."

(Dr. H. W. Spencer, St. Louis, Mo.)

"In lymphatic types, return; in about five per cent I believe we get some return." (Dr. Sherman Voorhees, Elmira, N. Y.)

"Seldom present if thoroughly done, unless we operate before the fifth birthday." (Dr. Henry L. Swain, New Haven, Conn.)

"Recur but seldom."

"No recurrence after complete removal."

(Dr. Charles F. Grayson, Philadelphia, Pa.)

"Yes, in small number of cases."

(Dr. J. Frank Crouch, Baltimore, Md.)

"No brutal thoroughness can prevent later growth of lateral adenoid tissue, if condition of stuffy nose is allowed to persist or return. Excess of excision can give pharyngitis sicea. Recurrence rare after fair removal and attention to nasal freedom." (Dr. B. Alexander Randall, Philadelphia, Pa.)

"Rarely." (Dr. Erastus E. Holt, Portland, Me.)

"If entirely removed, impossible."

(Dr. John Van Duyn, Syracuse, N. Y.)

"There is no recurrence after the removal of the adenoids 'in toto.' " (Dr. Richard Jordan, New York, N. Y.)

"Very unusual." (Dr. Hermann Knapp, New York, N. Y.)

"Exceptional." (Dr. J. H. Stucky, Lexington, Ky.)

"No recurrence if operation is complete."

(Dr. E. W. Fleming, Los Angeles, Cal.)

"Don't believe in recurrence, but have seen re-growth from incomplete removal." (Dr. Joseph A. White, Richmond, Va.)

"Exceptional but not infrequent."

(Dr. Henry W. Ring, New Haven, Conn.)

"In about one-sixth of cases, recurrence."

(Dr. W. Freudenthal, New York, N. Y.)

"Recur in small per cent of children when operated upon at an early age, two to five years."

(Dr. W. Harvey Smith, Winnipeg.)

"Recur, no matter what you do, in certain cases."

(Dr. Frank E. Miller, New York, N. Y.)

After careful consideration of the foregoing information, I think the following conclusions may be drawn:

1st. That the majority of physicians prefer to use a general anaesthetic when performing the adenoid operation.

2nd. That ether is used by far the larger number of men.

3rd. That most men prefer a light anaesthesia.

4th. Fully four-fifths of the operators favor some form of recumbent position.

5th. Most physicians insist on thoroughly exploring the nasal pharynx with finger at conclusion of operation.

6th. The majority use both forceps and curettes of different makes, but there is a tendency towards the use of curette alone.

7th. After treatment is a simple affair with most men, the important points being rest, light, diet, and the maintenance of nasal respiration.

8th. Recurrence is rare, after thorough operation, except in children with lymphoid tendency.

Finally, I wish to thank all those who so kindly answered the questions I sent them, and to apologize for not giving credit for replies in some cases, as the writers did not sign their names.

Discussion.

Dr. Edward B. Dench, of New York City, took issue with the reader of the paper upon the point of not removing adenoids and tonsils at the same operation, as the disadvantage of giving a child an anesthetic the second time more than counterbalanced the little additional hemorrhage which might be encountered.

Dr. Hiram Woods, of Baltimore, said someone was quoted as having removed the adenoid tissue from the fossa of Rosenmüller with a curette; he would like to see that curette. He had never seen anything but the finger that would answer this purpose. The question of recurrence deserves more attention than it receives. What is this recurrence? Is it part of the base that has not been removed, or is it cicatricial formation? He had seen cases

of what seemed to be recurrence of adenoid tissue, even after thorough removal. Just what the pathology of this recurrence is he did not know.

Dr. Robert C. Myles, of New York city, thought it interesting to get a general opinion upon this subject, concerning which the mental attitude seems so peculiar. A simple mechanical proposition should be agreed upon by all. About twenty years ago it was the habit in England and other countries to remove all the adenoid tissue as well as some of the mucous membrane. The rhinopharynx was cleaned out so thoroughly that there was little opportunity left for regrowths. That was the era of the finger and the forceps. Then came the era of the curette. In certain types of cases, it is impossible to engage all the adenoid tissue with the curette. The adenoid tissue often grows on the Eustachian tube, and at times forms synechia with the tissue of the vault. To remove this with the curette is impossible. Large masses can be removed with either forceps or curette, but those parts which grow in the fossa of Rosenmüller and into the posterior nasal fossa, must be removed with the finger and forceps, and the finger must be educated for the purpose. The chief objection to the curette is that it frequently removes quite a piece of the mucous membrane below the adenoid. If the patient is profoundly anesthetized —and after the tonsils have been removed—I prefer to remove the adenoids radically with the forceps, guided by the finger, and know of no other way by which it can be done. As to recurrence: In the child under two years of age, whose adenoid tissue is in process of development, with the basic part of the lymphoid tissue more or less buried in the mucosa, unless that basic part is removed the same thing will occur after the operation as occurred after its birth—development of new adenoid tissue. In this class of cases, under repeated inflammatory conditions, there is hypertrophy and certain amount of recurrence.

Dr. Wendell C. Phillips, of New York city, agreed with Dr Myles concerning the cause of the recurrence in very

young children. He did not agree with him and others with reference to adenoid tissue in the fossa of Rosenmüller. He always makes a careful examination, even in young children, and he very rarely finds adenoid tissue arising from this locality. He always makes an examination with the mirror after the operation. He could not recall a case in which the growth was situated in the fossa of Rosenmüller. The overlapping of the main mass and the inflammatory condition which arises therefrom is the real condition in these cases. He considered it a mistake in infants to attempt to do a thorough operation because of the danger of tearing the mucous membrane from this area. He preferred to remove enough of the mass to relieve the child during the nursing period, and to remove the rest at a later date.

Dr. James Kerr Love, of Glasgow, Scotland, said that in adenoid operations it is the "man behind the gun" rather than the particular instrument which he uses, that makes for efficiency. The operation should be considered a serious one, and the patient should be kept under observation for a day or two afterward. The question of recurrence must be faced by everyone from time to time. The deafness which remains after operation is often due to the obstruction of the middle nose. If the middle nose, (the middle and inferior turbinates) were operated upon in these cases of adenoids in which thorough operation has been done there would be less talk about recurrence of the adenoids themselves, and there would be greater improvement in hearing.

Dr. George A. Leland, of Boston, called attention to the fact that at the International Otological Congress, at London, in 1899, Rudlolf, of Wiesbaden, stated that from 3½ to 4% of adenoids recur. That was the opinion of the Germans then, and he did not think they had changed their opinion up to the present time. Why do they recur? In the first place, it is an anatomical impossibility to remove all of the adenoid tissue. Dr. F. H. Hooper, in 1883 or 1884, first introduced the operation in Boston, and he insisted that they be absolutely removed. He was shown, at the Harvard

Medical School, that it was absolutely impossible to do this on the living, because at the top of the vault, near the septum, there are small bony sulci, clefts, and fissures, in which the mucous membrane is reduplicated, and in these reduplications the lymph glands are attached. It would therefore be impossible, except on the dead even with direct illumination, to remove them all.

Someone said adenoids do not grow in the fossa of Rosenmüller; by means of the eye on his index finger, and the eyes in his head, he knew that they did grow in the fossa of Rosenmüller as well as around the opening of the Eustachian tube, and that it is very difficult to remove these. He devised and used an instrument, medium sharp, made by Hajek of Vienna, in 1882, which goes up into this fossa. He had said little about it because of the danger to the Eustachian cartilage.

Some years ago, in 1898, when the American Laryngological Association met in Brooklyn, it was brought out by the speaker, that one cause of adenoid hyperplasia is mouth respiration; therefore, if nasal respiration is not established after removal of the adenoids, the same force is operative. Both parents and physicians are at fault, if they do not see to it that the nasal respiratory habit is restored. It does not restore nasal respiration simply to remove the adenoids.

He thought it desirable to remove adenoids and tonsils at the same time.

Dr. B. Alexander Randall, of Philadelphia, insisted upon the use, both before and especially after operation, of silver nitrate, disagreeable or brutal as it may be, since there is usually much lateral thickening which may be wholly unrelieved by the operation, as Meyer himself pointed out.

Dr. Phillips called attention to the Stubbs curette, first brought to his notice by Dr. Thomas J. Gallaher, of Denver. It gets well up behind the growth and is the best curette in existence for the removal of adenoid growths. Dr. I. I. Thompson had devised a protector which slips over the curette and prevents its getting dull.

Dr. Frederick L. Jack, of Boston, called attention to the danger of large detached pieces of adenoids in the throat. He had been taught to use the forceps by Dr. Franklin Hooper, of Boston, who was one of the first to call general attention to the operation. He has seen large pieces of adenoid tissue disappear from the curette, frequently swallowed by the patient. The protectors on the curettes are not always satisfactory. He preferred to do the operation with forceps and smooth the edges off afterwards with the curette. Forceps will hold on to the pieces removed, and will remove a growth of any size, whereas the curette will not take in the entire growth. The chief objection, however, to the curette, is the possibility of the adenoid getting into the trachea; a second objection is that it is not quite as thorough as forceps.

Dr. Dench used nitrate of silver a good deal after operation, and had found that it aided materially in the prevention of recurrence. The application is made through the anterior nares, after shrinking the tissues by means of cocaine or adrenalin, sixty grains to the ounce of nitrate of silver being directed backward to the base of the adenoid. It is a relatively painless procedure. The application is made directly to the cut surface which represents the base of the growth removed.

Dr. W. C. Braislin, of Brooklyn, referring to Dr. Randall's advocacy of silver nitrate, said that in making applications of silver nitrate to the pharynx, he had tried to get the applicator within or to the mouth of the Eustachian tube. This can be accomplished by drawing the applicator, introduced into the naso-pharynx, well forward, from one side to the other, fairly well against the mouth of the tube. It produces good results in young adults and children. The fossæ of Rosenmüller may be curetted several times with the fingernail. This is of advantage in children who show symptoms of impaired hearing when there is reason to attribute it to blocking of the tube.

Dr. George E. Shambaugh, Chicago: The impression has been given by some of the speakers that whenever an operation for the removal of adenoids is undertaken the faucial tonsils should also be removed. This is about the same as saying that in all cases faucial tonsils should be removed since adenoids may develop necessitating an operation independent of any symptoms of the faucial tonsils. He sees no reason for subjecting a patient to an operation on the tonsils that are not enlarged, have never produced any symptoms that would warrant a diagnosis of disease of the tonsils. In cases of this kind where the adenoids are enlarged and require removal, this operation can be performed by the use of curettes entirely satisfactory with a gas anaesthesia, making the operation a very simple one, whereas, a tonsillectomy is always a more or less formidable operation when properly done. He does not approve of the indiscriminate removal of faucial tonsils without adequate indications.

Dr. Smith, in closing the discussion, said he thought the pediatrist must modify the views of the otologist with reference to the cases under discussion. It is for the pediatrist to see that the child is in good condition before undergoing such an operation as removal of adenoids and tonsils, as it is he who looks after the patient after operation. There is a tendency among many to delay removal of adenoids until the child is five or six years of age, unless it is losing ground, because the probability of recurrence is not so great after that. The ideal forceps, in his opinion, are those of McAuliffe, of New York.

OSTEO-MYELITIS OF THE TEMPORAL BONE.

CHARLES W. RICHARDSON, M. D., Washington, D. C.

On November 12, 1903, I presented a paper before the Section on Otology of the New York Academy of Medicine, on the subject of Acute Osteo-Myelitis of the Temporal Bone. Since writing the above-mentioned paper, I have not encountered another case of this unusual form of suppurative inflammation of the temporal bone until this winter. This case lacked the marked local symptoms of osteo-myelitis, that is, the extensive area of local tenderness, but presented in a marked degree all of the profound constitutional evidence.

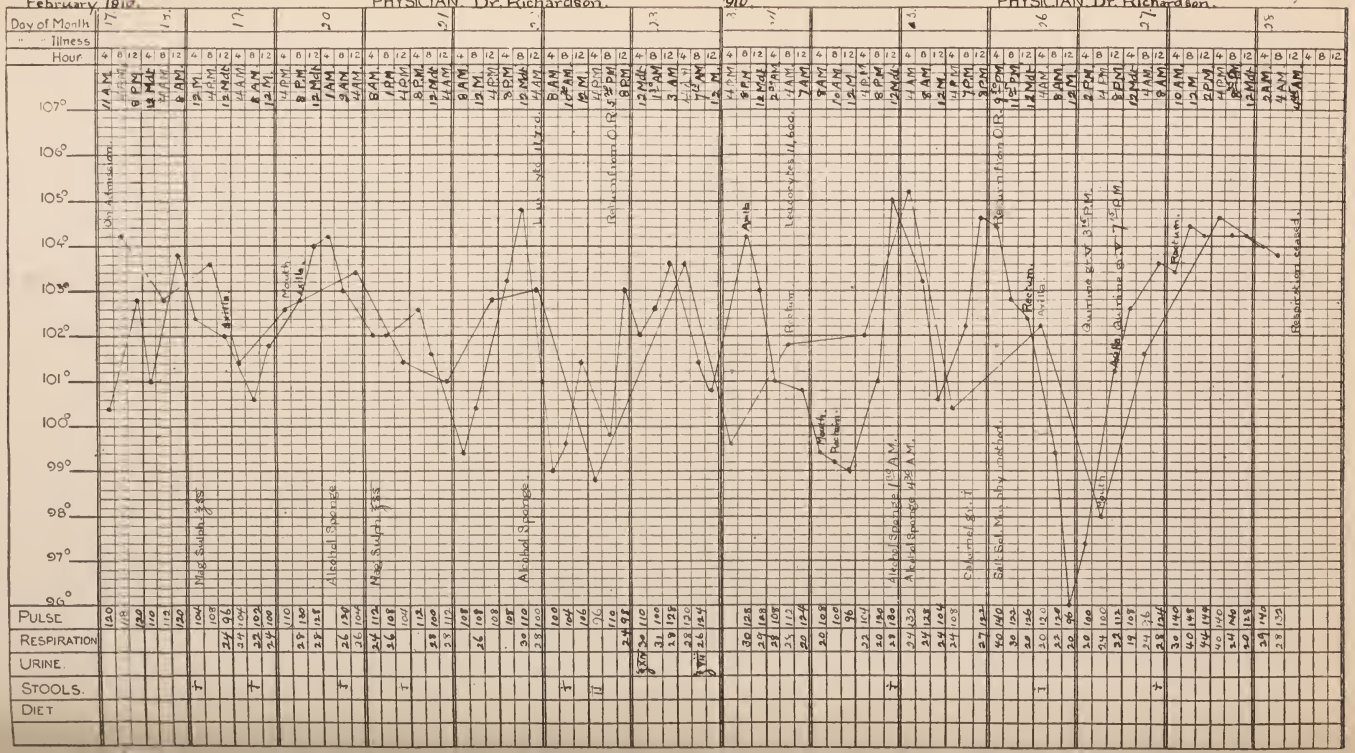
A young boy of ten years of age was admitted to my service at the Episcopal Eye, Ear and Throat Hospital, February 17th, with the following history: He was an inmate of the Industrial Home School. Three weeks before his admission to the hospital he had had a severe attack of acute bronchitis, from which he was convalescing, when he was seized with sharp pain in the left ear. On the third day following the initial ear symptoms he was brought to the hospital. I saw the patient shortly after his admission. He was a pale, fragile child, under-size and under-weight for his age. He had the appearance of being quite sick; quite intelligent, non-complaining, and very quiet. On examination of his ear, I found the membrane to be very vividly congested and bulging, necessitating immediate opening. The temperature was 103° at this time. The exudation from the middle ear followed the usual course, but was never abundant. The temperature showed a distinctly fluctuating character for the following four or five days. The patient had the appearance of a very sick child, kept very quiet, talked very little, and never complained; quickly complying with every request made of him. There were no mastoid tenderness until the fourth day, when firm

pressure over the antrum and at the tip of mastoid caused evidences of pain. A leucocyte count gave 11,700 white cells. On the fifth day, February 22, the mastoid tenderness now being very great and extending over the whole area of the mastoid, the operation was immediately performed. It was noticed that the cortex had a peculiar bluish-white appearance. On chiseling through the cortex into the cells, the peculiar friable nature of the bone was noted; there was an absence of the brittle firm feel of the healthy cortex. The mastoid contour had a grayish-white appearance which is spoken of as the shaven beard appearance, and all the small cells were filled with pale granulation tissue. There was no pus demonstrated anywhere, not even in the antrum. That peculiar characteristic of osteo-myelitic bone imparting the sensation as though you were cutting through wet blotting paper was maintained throughout the mastoid. It seemed almost impossible to reach firm, hard, healthy bone in any direction. The patient was returned to ward in good condition. The following day found the patient in about the same condition, with the temperature showing a more distinctly septic wave. Blood count demonstrated 100 less white cells than on the previous count. Drs. Ackes and Wood were carefully following the case from the internist's point of view. They could find nothing to account for the continued sepsis and were strongly inclined to believe that the lateral sinus was infected. This did not coincide with my views. On the evening of the 25th, unwillingly, I decided to expose the sinus. On exposure, the sinus appeared normal, but in order to have no uncertainty upon this point, I opened the sinus, but found no evidence of clot. On the 27th, the patient developed pneumonia and died on the morning of the 28th of February, eleven days after his admission to the hospital. Autopsy was made by Dr. L. Z. Glazebrook. The autopsy findings demonstrated changes in the organs in which diseased conditions had been recognized before death. There was evidence of a recent pneumonia in the upper lobe of right lung and a plastic exudate

EPISCOPAL EYE, EAR, AND THROAT HOSPITAL.

NAME Frank Miskell. DIAGNOSIS. Mastoiditis.

February 1910. PHYSICIAN. Dr. Richardson.



ISCOPAL EYE, EAR, AND THROAT HOSPITAL.

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910. PHYSICIAN. Dr. Richardson.

in the pleura of the left side. The brain and its membranes showed no pathological change whatsoever. The most interesting autopsy manifestation was in connection with the temporal and sphenoid bones, the temporal bone throughout the whole pyramid presented the same condition as had been noted in the operation on the mastoid. It was so soft and friable that it was found impossible to remove it in its entirety. The sphenoid bone presented on the corresponding side the same identical condition as the temporal bone.

Discussion.

Dr. Hiram Woods, of Baltimore. would like to hear from some of the members with large surgical experience concerning their observation upon this purely exploratory incision, with no positive evidence of the disease. He had seen a man in Baltimore do that. The hemorrhage was easily controlled, but there was marked prostration afterward, and the patient died of what was pronounced septic pneumonia. He did not know whether the pneumonia was in any way to be associated with the exploratory incision.

Dr. Edward B. Dench, of New York City, would hesitate to open the sinus if it is absolutely normal on the outside. He had done it in one or two cases for exploratory purposes. In cases where there is sinus thrombosis the clot may be parietal. Ordinarily the outer covering of the sinus gives some evidence as to whether or not there is a clot. He would have opened the sinus in the case reported by Dr. Richardson. Where one is in doubt and there is nothing else to explain the temperature, the sinus should be opened.

Dr. B. Alexander Randall, of Philadelphia, said the question of the sinus is a little aside from the subject under discussion. There are many cases of involvement of the sinus that do not go on to thrombosis. While an exploratory incision may be regarded as harmless, it is doubtful if the

thrombus can always be recognized after the incision is made. The long incision necessary to determine whether there is a parietal thrombus or not must be left to other hands, when the absence of septic indications leaves it improbable that there is more than a phlebitis.

Dr. Richardson, in closing the discussion, said one of the most interesting features of the case reported is the peculiar chart, and the fact that the child was profoundly ill. The condition of the temporal bone was quite characteristic. He gave a positive opinion that it was a case of osteomyelitis, and saw no reason for changing it throughout the whole course of the case. When the internists and others insisted that the sinus was involved, thinking that they might be as nearly right as he was, he decided to take their advice and open the sinus. He did not care to do such things, and did not make a practice of opening the sinus, but in this case it was justifiable. He had hoped to get the bone in its entirety and to present it at this meeting, but it was impossible to remove it as a whole because it crumbled under attempts to remove it.

STUDY OF SOME CASES OF ACUTE OTITIS MEDIA, SEEN IN CONSULTATION.

1. PRIMARY OTITIC INFECTION.
2. DELAYED APPEARANCE OF OTITIS.
3. PROLONGED REMITTING FEVER. DIAGNOSIS.
4. CONVULSIONS.
5. REFERRED PAIN.

By HIRAM WOODS, M. D., Baltimore, Md.

If the better class of general practitioners in Baltimore is typical of their associates in other cities, it is evident that appreciation of early ear troubles is keener than it was a few years ago. Physicians are learning that the welfare of the ear or, possibly, the issue of life and death, depends on the diagnostic acumen of the man who first sees the case. The lesson which specialists have been trying to impress for so long is at last being taken home.

During the past few years consultations regarding a possible otitic origin of systemic conditions have greatly increased. These consultations have developed a number of facts which, I have thought, might furnish a basis for profitable discussion; hence the presentation of so trite a subject as acute otitic media.

1. Primary Otitic Infection. General attitude toward otitis media, taught in text books and unquestionably largely correct, is that the ear becomes involved as a sequel to infection elsewhere. Among these infections is that great waste-basket, grip. After a few days of fever, malaise, with no definite symptoms of anything, the patient, if a little child, puts the hand to the ear, thus calling attention to it, and examination reveals otitis media. Again, there is complaint of pain, followed in a few hours by discharge; or otorrhoea is the first observed ear symptom. Diagnosis of grip, with secondary otitis media, is satisfactory to the physician, and maybe to the aurist, too. It was to me until the care of

some medical friends in diagnosis taught me otherwise. To justify the diagnosis of primary otitic infection we would have to find: 1, systemic evidence of infection; 2, objective evidence of ear disease; 3, absence of all local or general disturbances capable of producing ear complications; 4, relief of constitutional symptoms and restoration of health by treatment confined to ear drainage. Pain is such an overshadowing symptom of otitis as to be practically the only one of which the physician takes cognizance. But pain in a child, too young to tell about it, is easily overlooked. Another still more significant thing is that pain is by no means invariably present in otitis media. Hence, if primary otitic infection is an entity, if it is capable of producing systemic infection, it is our duty to urge on the internist the necessity of including the ear in routine examination. How the infection reaches the ear without manifesting its presence elsewhere is interesting, and possibly, not difficult of explanation. It is well known that infectious organisms of different kinds, virulent and innocent, stay in the nose and naso-pharynx for a long time, producing no results. They could easily reach the tympanum and manifest themselves there. Again, it is conceivable that they could come through the circulation.

The important clinical feature, however, is that this otitic infection does not always show itself in the orthodox order of exciting cause, pain and other subjective symptoms before the discharge makes diagnosis certain. Without them nothing but routine examination of the ear, by a physician who has qualified himself to make it, can demonstrate the cause of general illness. If he appreciates the meaning of injection of malleus vessels, redness of Schrapnell's membrane, or even knows that the ear can be primarily infected and calls in a consultant, the diagnosis is easy. In a number of cases with such symptoms and objective evidence of otitis media, I have found slight impairment of hearing, lateralization by Weber's test, and have seen recovery follow incision of the membrane. In one case last year, a boy of ten, without pain, a temperature of 104°, and a slight redness and bulg-

ing of the supero-posterior angle of the membrane, a single drop of streptococcus pus was evacuated, followed after a day by discharge. Symptoms were dispelled by the incision.

Akin to primary otitic infection are the delayed ear complications of various diseases. I allude to otitis media, sequel of infectious diseases, but removed from the causative disease long enough to throw doubt upon causative relation. Either there is latent tympanic infection after the acute disease, or a new ear infection. For instance, a child has measles and passes through the attack without ear symptoms. Two or three weeks later, without other symptoms, and when recovery is seemingly complete, otitis media develops. Such cases I believe are not particularly uncommon, after measles and grip. In a few which I have seen there was the typical order of pain, fever and otorrhea, unless the case was seen before perforation. In other cases, sudden return of fever was the only symptom, and otitis was found because the physician looked for it or got another to do it for him. Latent infection of the middle ear shows itself, too, in another way: apparent recovery of acute otitis media, with late mastoid symptoms. I brought this subject before the Otological Section, of the A. M. A., in 1901, and have seen several cases since. One will suffice. A man of 48 years, after exposure in riding in the front seat of an electric car, developed pain in the right ear, with a temperature of 102°. He was seen by his physician who found nothing but fever and ear pain. The drumhead presented all the indications for incision, but operation was refused. Two days later the physician reported that ottorrhea had appeared, with relief of pain and fall of temperature. Soon I learned that hearing for the watch was normal, and the patient entirely recovered. He went along with his business for seven weeks, when he was seized, while at his office, with severe earache. Mastoid symptoms developed rapidly, and on operation after twenty-four hours the antrum and cells were found necrotic and full of pus. The easiest explanation seems to be that infection went to the mastoid at his first illness; but why it was so long in causing symptoms, or why

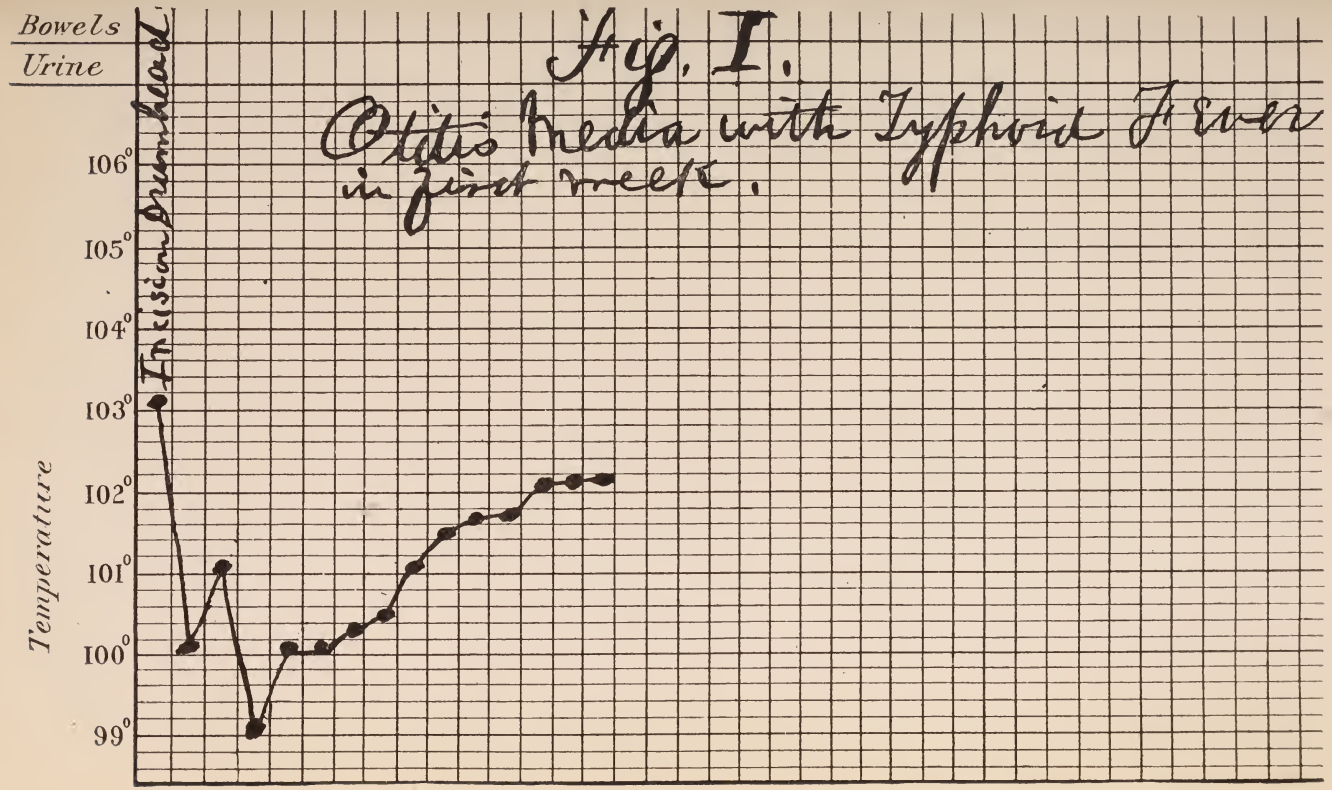


Fig. I.
Otitis Media with Typhoid Fever in first week.

he was apparently well for seven weeks, with disease progressing in his mastoid, is not so easy to account for. Such cases have one important bearing on every day otology. I believe we all see cases of deafness following infectious diseases without history or evidence of inflammatory otitis. There is nothing in the patient's condition to account for it. Fork tests show it is due to middle ear disease. Inference seems justifiable that the acute disease left otitic infection not sufficiently virulent to cause immediate symptoms, but capable of doing one of two things: either slowly breaking down tissue through spread of infection, or keeping up a constant hyperaemia, leading to exudate, and so bringing about sclerotic changes in the tympanum. One of the facts we should try to impress on general men is that these latent ear infections are frequent: that their ultimate results are uncertain: that the tympanum is in the natural path of danger just as the lungs are, and that a case should not be dismissed as well until adequate ear examination has been made. Men should know from us that this is the time to save hearing.

A feature which often puzzles not only the physician but his consultant is the meaning of prolonged fever in a seemingly light case of otitis media. During or after an infectious disease, otitis media develops. Maybe the membrane is incised: again it is allowed to ulcerate. The original trouble recovers. So does the ear complication, to all appearances. Pain disappears, the discharge lessens or ceases, hearing improves. Yet fever continues. If due to the ear, it is our duty to secure complete drainage: if not, the physician wants to know. It is a question of diagnosis which may, on the one hand, involve mastoid operation, not for mastoiditis but for drainage, and at the same time we must be careful not to operate on a patient for ear sepsis when he has something else. Brief notes of three cases are appended:

Case 1. A man of 31, developed acute otitis media after rhinitis. Pus after membrane incision. Two days later, mastoid tenderness. Temperature for the first week is given

in Fig. 1. On the fourth day he was sent to the hospital as mastoid operation was thought probable. Local symptoms were less pronounced the day after, and almost disappeared in another twenty-four hours. For the next three days he ran a steady fever, without remissions. His physician, Dr. Charles W. Mitchell, was unable to determine its cause, if the ear and mastoid were excluded. Three factors in the case led me to do this: 1, steady improvement in local symptoms, and their total disappearance after a week; 2, character of the fever line; 3, a leucocytosis of 4,000. Typhoid fever was thought of; but it was the 16th day before rose-spots were found, and positive Vidal was obtained only toward the close of the fourth week.

Case 2 shows how easily one can overlook a concurrent lesion. A healthy, athletic man of 29, without previous illness, was attacked early Saturday morning with a chill and right earache. He fought it out alone all day, but on Sunday called in an aurist, who found temperature of 104°, with objective evidence of acute suppurative otitis media. For reasons I did not discover, he postponed myringotomy until Monday afternoon when it was done at a hospital under chloroform. To Wednesday night temperature ranged above 103°, without remission. Farther operative procedures were then considered; but a consultation was asked by the family. I saw the case Thursday afternoon. That morning, six days after the initial chill, temperature had dropped to 97.5°. I found the ear dry, and no evidence of mastoiditis. The outcome of the case was that the man was allowed to continue an uninterrupted recovery from an unrecognized, limited, right pneumonia.

Case 3. Temperature chart of a third case (Fig 2) is given by way of comparison. The patient was a girl, 12 years old, who developed left acute otitis media, after apparent recovery from uncomplicated measles. She had been kept in the house and for the most part in bed for the eight days before ear trouble was found. Her physician, Dr. Mitchell, found it in routine examination for relapse of fever. She had nothing directing special attention to the

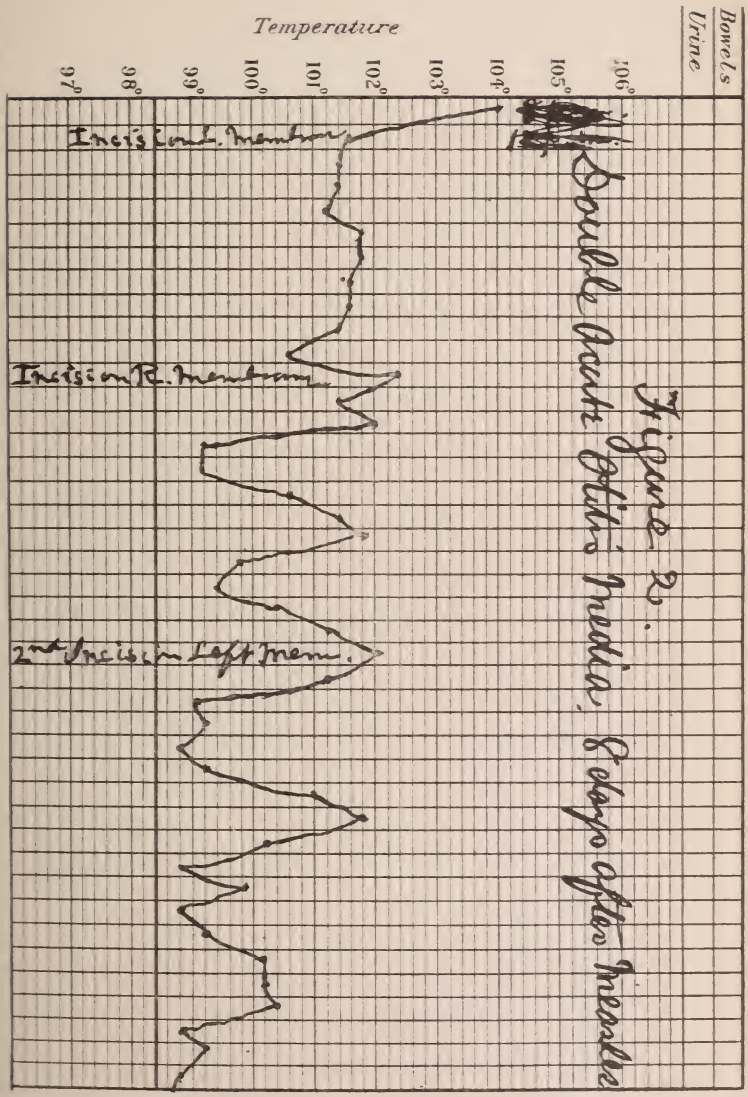


Figure 2.
Double Acute Otitis Media. 8 days after measles

eàr. When called in, I found definite left suppurative otitis, without perforation, while the drumhead of the right was congested. Leucocytosis was 16,000. After left incision, which I did at once, temperature fell from 104° to 101.6°, and stayed there two days. Then the right drumhead was incised, though there was no pain. For the next five days there was remitting fever. On the fifth, the left mastoid was tender to pressure, but no pain otherwise. I asked Dr. Harry Friedenwald to see the case with me, and we decided that symptoms were not sufficiently urgent for mastoidectomy, and that the left drumhead, the one first incised, which had healed, should be incised a second time. After this, mastoid tenderness disappeared, and the recovery, though slow, was uninterrupted.

The case illustrates: 1, the delay which may ensue after disappearance of the causative disease before ear complications develop; 2, what I believe to be the characteristic of otitic temperature, in all essentials, that of a mild sepsis; 3, the value of leucocyte count. When able, I have this count made in acute otitis media, and it rarely is below 15,000. Considerable observation of cases like these has led me to think that in the temperature line and leucocyte count we have pretty reliable guides regarding an otitic cause of systemic disturbances as distinguished from essential fevers. There is always the possibility of another lesion with the ear trouble.

There is another side to this matter. Sometimes we are compelled to do a mastoidectomy for drainage, though there are no symptoms of mastoiditis. Even when the process is painful on pressure, tympanic drainage is often enough. In deciding if mastoid drainage is needed, I believe the amount of the otorrhoea is an important guide. There is nothing new about this; yet it is a guide often neglected. It is unnecessary to relate cases in which, without definite mastoid symptoms, operation has been done because of persistent fever, of septic type, and profuse purulency. Chills, sweating, extreme variations in temperature, (105 and upwards, to subnormal) profound exhaustion—the things

which lead us to uncover the sinus,—are absent. The cases drag themselves along for weeks, with about such a temperature line as given in case 3, an abundant purulent discharge, and no pain. When they come to operation, extensive disease is found in the mastoid walls. A question I should like to hear discussed is: What is the line of demarkation between the safety of tympanic, and necessity for mastoid, drainage? Is there anything, outside of time and profuse discharge, to guide us?

I want, finally, to call attention to three cases of acute otitis media, which were new observations to me. The first is that of an infant, five months old, whom I saw in consultation with Dr. Thomas R. Brown. At the age of three months the baby had measles, which left in its wake enlarged cervical glands. The week before I saw her, she had a transient, general vesicular eruption which Dr. B. diagnosed as chicken pox, Three days after disappearance of eruption, there was a convulsion, followed by stupor, lasting an hour or so. Rectal temperature was 103°. Dr. Brown found a leucocytosis of 18,000 at his first visit. While in the house the child had a convulsion, with dilated pupils, rigidity, and unconsciousness. He examined the ear, and found, as he thought, redness and bulging of the sub-post. angle of the membrane. I was called in at night. Temperature then was 100, barely, the child was free from pain, took its bottle eagerly, and did not look sick. The right drumhead presented the evidences found by Dr. Brown in the morning; but there was no bulging, and in view of the generally improved condition, I decided to do nothing till the next morning. At quarter of nine a. m., I received a phone message that the child was worse, had had two convulsions, and rectal temperature was 103°. As soon as possible Dr. Brown and I reached the country home of the patient's parents. The drumhead was now bulging in the sup-post-angle, and leucocyte count was 24,000. Without anaesthetic I incised the drumhead, continuing the incision into the canal wall. A very little pus was evacuated, and recovery was uneventful. While this is my one observation of

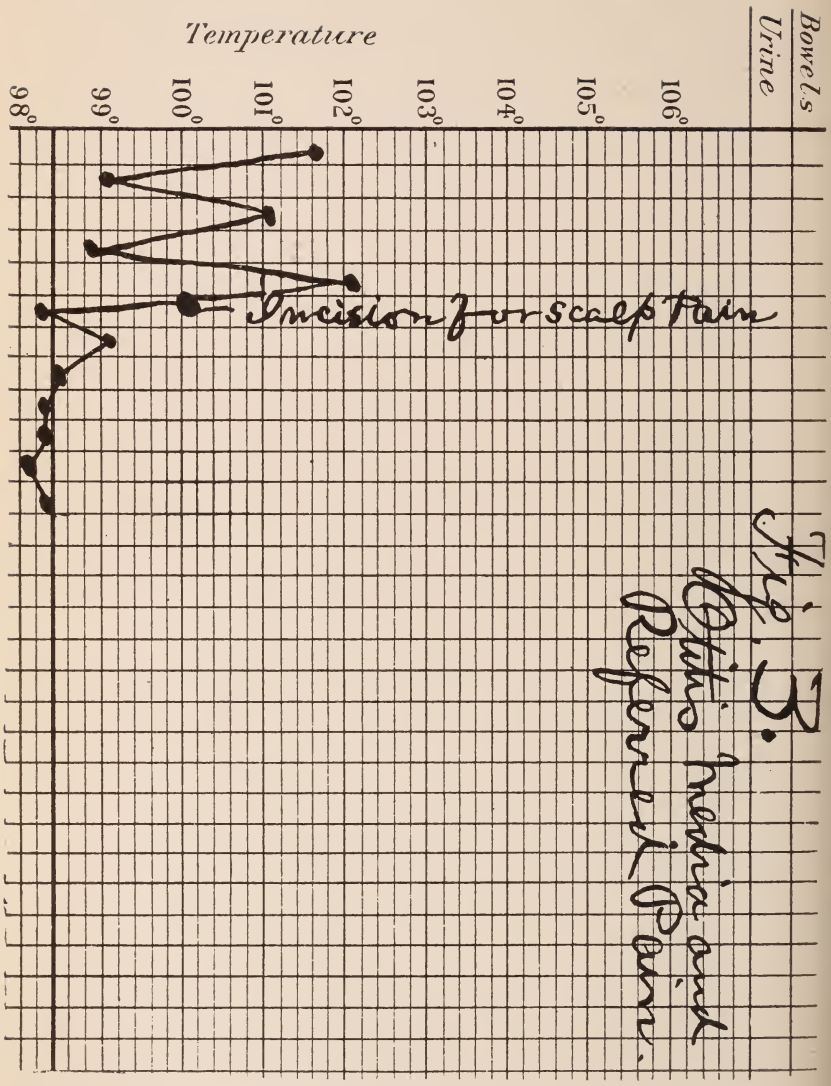


Fig. 3.
Otitis media and Referred Pain.

ear convulsions, I have known, as have all of us, of babies dying of supposed meningitis, with otorrhoea toward the close. I can count a half dozen such cases in the families of friends and in conversation with physicians. The intimate connection between the tympanic and cranial cavities in infancy, through the squamo-petrosal suture, is probably sufficient explanation. The interesting fact, however, is that physicians are appreciating such possibilities.

The other two cases I have classified as "Referred Pain." The first was a nurse, Aet. 24, at the Union Protestant Infirmary, who came to me on Jan. 25th last with a history of left peri-auricular discomfort for two days. The night before I saw her, she was awakened by severe pain along the anterior border of the sterno-cleido-mastoid muscle, at least two inches below the tip. The slightest pressure here over an area of an inch in circumference was painful. Temperature was normal and no hearing defect could be found. The drumhead presented a normal appearance. Leucocyte count was 8,000. She was put to bed, and given salicylate of sodium, and later aspirin, with little effect. Morphia was needed to secure relief from the strictly localized pain. The only pain in the ear proper was produced by brushing with a cotton pencil or touching with a probe, the posterior wall of the canal. Hearing for the watch gradually diminished, and in four days was only on contact. A loud whisper could then be heard at two or three feet, and fork test, Weber's, was lateralized to the left ear. I could find nothing by inspection, save, possibly, increased blanching of the drumhead. Temperature during these five days did not go above 99.5°. On Jan. 31st, on account of the falling off in hearing, I determined to incise the drumhead, which was done under nitrous oxide. The membrane cut like stiff parchment. Through the lips of the incision there escaped a few thin flakes looking like very thin bits of paper. They were lost in a drop of clear fluid which followed them and the little blood from the upper edge of the incision. The next day she heard the watch at six inches, and in a short time had normal hearing. Twice after in-

cision, on Feb. 1st and 2nd, temperature reached 101°. This followed an attempt to syringe the little clot from the canal. The pain in the muscle was relieved at once by the incision of the membrane.

The other case occurred in a man, a lawyer, whom Dr. Brown asked me to see with him. Mr. W., 49 years old, was first seen by his physician on the morning of March 21st for a slight right tonsilitis. It subsided after two days. Dr. Brown was summoned again early in the morning of the 24th for severe pain in the right parieto-occipal region. As seen by the chart, Fig. 3, there was a temperature of 101.6°. I saw Mr. W. on the morning of the 25th. A mustard plaster had been applied before Dr. B.'s visit, and the scalp was reddened. Pain came in paroxysms, the slightest movement producing it. It was confined to a limited area, at least two inches behind the meatus, the intervening space over the mastoid being free from tenderness or congestion. The drumhead presented the appearance of a mild catarrhal otitis. Hearing was normal for watch and speech. Because of the remoteness of pain from the ear, and normal hearing, I hesitated to connect the ear with it. He was given an atrophia and cocaine solution to use during the day, but reported in the evening that he had no relief. His temperature was 102°, while the drumhead presented the same appearance as in the morning. Weber's fork test was lateralized on the affected side. In spite of morphia he had a wretched night, every movement of the head causing paroxysmal pains limited to this one spot in the scalp. I saw him again at 8 a. m. on the 26th. He was prostrated by two sleepless nights, and accepted what I told him was a purely experimental incision of the reddened sub-posterior angle of the drumhead. His hearing was absolutely unaffected save for Weber's lateralization. Fair anaesthesia was obtained by an alcoholic and oily solution of cocaine (20%). Relief from pain was almost magical. As in the case of the nurse, the drumhead cut "stiff." There was some little hemorrhage, but no pus, so far as we

could see. Recovery was prompt without impairment of hearing.

I presume these referred pains were due to an unusual connection between the tympanic nerve and other branches of the glosso-pharyngeal through the petrosal ganglion.

I have thought these observations in acute otitis media of sufficient interest to put on record. Behind them, however, is the fact that but for the thoroughness of diagnosis on part of the internist, the cause of fever, convulsions, pain, would not have been determined. Improved attitude toward ear troubles on part of our associates has brought a definite change in our practice, at least in mine. In private work chronic suppurative otitis media, of which years ago I saw so much, is rare. Even acute otitis media, allowed to go on to ulceration of the membrane, is not very frequent. But constitutional symptoms due to ear disease, without gross external evidences of the latter, are more common. The moral of it all is that aurists have got to be good enough doctors to make differential diagnoses.

Discussion.

Dr. E. Terry Smith, of Hartford, Conn., had employed crystals of carbolic acid menthol and cocaine rubbed together as an analgesis in opening the drum membrane. It produces complete analgesia of the drum, and at the same time disinfects the canal. He has seen no caustic effect, and it has been used by him with great success.

Dr. Edward B. Dench, of New York City, was interested in the cases in which the mastoiditis came on late. He had reported a case some time ago in which he opened the mastoid three months after the primary otitis. The patient was a man seventy-three years old who consulted him on account of defective hearing. There had been very little pain at the time of the initial attack of otitis, and only a slight discharge, which had disappeared at the end of the 48 hours. When the patient was first seen by Dr. Dench there was sagging of the postero-superior canal wall, and

this was the only sign present. About a dram of pus was discharged when the mastoid was opened. Another case, a woman between fifty and sixty years of age, was under observation for a month. There was absolutely no discharge from the ear when he first saw her, and no bulging of the drum membrane. One morning, after being under observation for a month, she came in with post-aural swelling. Upon operation considerable mastoid involvement was found. He believed that the mastoid infection in these cases takes place at the same time as the middle ear infection, there being a focus in the mastoid co-existent with that in the middle ear.

Dr. Ewing W. Day, of Pittsburg, was interested in the delayed infection. This had been characteristic in his section this year in grippe infections. The grippe would start off with ear infection and mastoid tenderness; this would subside, and the mastoid would give trouble a month or so later. That had been true in nearly all grippe cases this year, and likewise in exanthematous cases, especially scarlet fever and measles. He had recently operated on a case of scarlet fever in which the symptoms had entirely cleared up. The typhoid fever chart presented was characteristic of the normal condition. He and Dr. Jackson had rarely got mastoid involvement before the twenty-first or twenty-fourth day.

Dr. Woods, in closing the discussion, cited the case of a woman who came into his clinic with a little opening at the end of the mastoid. She had an old otorrhea of about twelve years' standing. He thought it was an old Bezold abscess which had not healed. She had been to two or three hospitals where she had been told that she did not need a mastoid operation because of the absence of symptoms. He used the von Pirquet test, which was negative; the X-ray showed nothing. He operated without any symptoms whatever, and found the whole thing absolutely necrotic.

A CASE OF FATAL MASTOIDITIS, SHOWING NO CLASSICAL SYMPTOMS FOR 13 DAYS BEFORE DEATH. DEATH DUE TO SECONDARY THORACIC COMPLICATIONS OF MASTOIDITIS.

By W. SOHIER BRYANT, A. M., M. D., New York, N. Y.

This case is a proof that mastoiditis may exist and result fatally without showing definite classical symptoms at the time of death. Although the conditions in the ear were apparently mild for 13 days before death, the primary cause of death was mastoiditis, with metastases of purulent bronchitis and acute pericarditis.

CASE. A woman, 63 years old. The patient was seen in my consultation service at the Manhattan State Hospital.

February 4, 1910. Patient was committed for chronic alcoholic insanity. Temperature, 98.4°; pulse, 80; respiration, 20. Physical condition good, except for otitis media of right ear. Slight bloody discharge from the right ear. Patient very deaf.

February 5. Temperature, 99°; pulse, 80; respiration, 20. Headache in vertex region. No vertigo, no nystagmus, no hyperesthesia or other pains. Patient dull and stupid. Left pupil slightly irregular.

February 6. Much nausea.

February 12. Sharp pain in right ear just before the external re-appearance of a discharge.

February 13. Sudden rise of temperature to 102.8°. Watch heard in right ear 2 inches. Watch heard in left ear 3 inches. I saw the case for the first time this day, and my examination disclosed acute otitis media of the right ear, an exacerbation of the chronic condition. There was considerable mastoid tenderness, and edema of the canal. The canal was closed by otitis externa. There was a wide perforation of the right tympanic membrane. The profuse

discharge from the right ear contained much blood and a small amount of pus. With these very definite symptoms indicating the presence of mastoiditis in the right ear, I advised that a mastoid operation be performed as soon as the consent of the patient's relatives was obtained. As treatment for the right ear, I advised irrigation with boracic acid solution.

The examination of the left ear showed an opaque flat thick membrane, with pink blush on the handle of the malleus. I did not hear of the case again until autopsy.

The following clinical notes were obtained from the hospital records.

February 14. The swelling had increased behind and in front of the ear. Discharge more purulent. Severe headache continues.

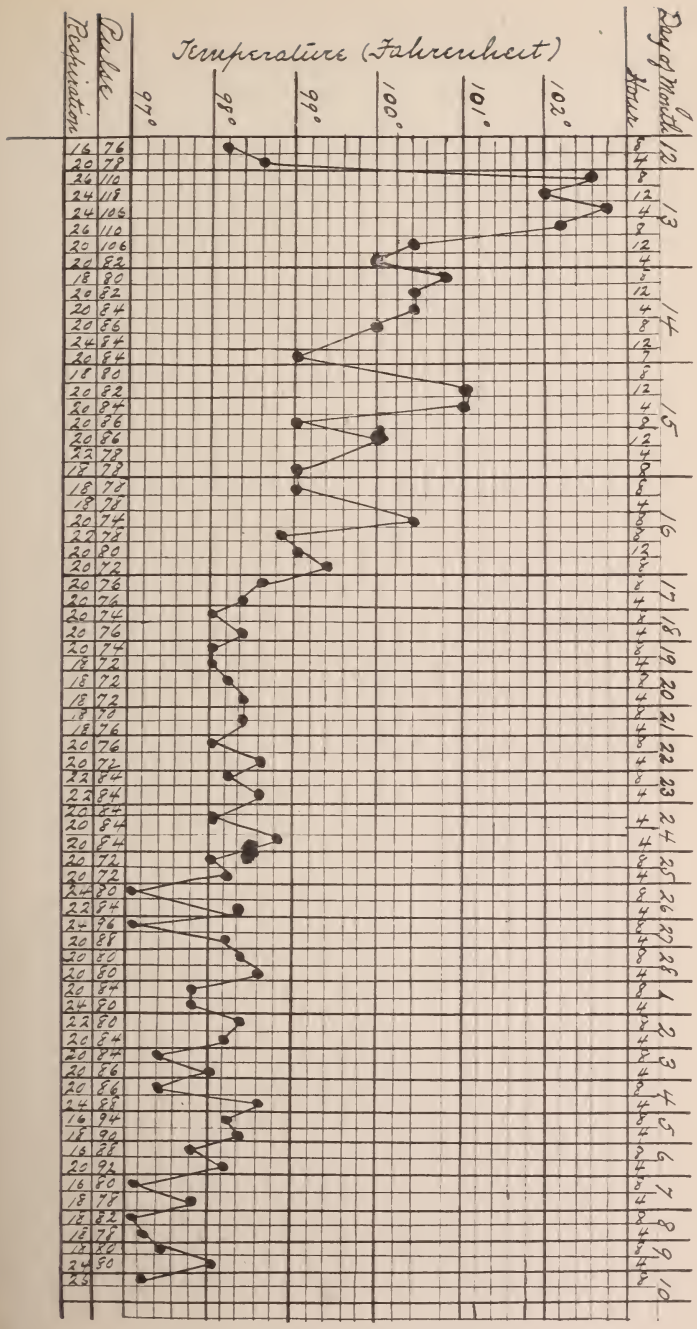
February 15. Sleeps most of the time. Deafness increased. Patient dull and apathetic.

February 16. Pain in right ear. Mastoid tenderness and edema extending down over the neck. Some redness in front of the ear. Moderate purulent discharge. Temperature, 100.4°; pulse, 90; respiration, 18.

February 25. Tenderness and swelling about the right mastoid and cervical region has now entirely disappeared, and there is only a slight discharge from the ear. The patient has no complaints. The irrigation is continued. The acute symptoms of the right ear having now subsided, the case appeared to be one of mild middle ear inflammation. The case did not now seem to demand an operation.

From February 25th to March 10th, the only symptom of disease, other than weakness, was a subnormal temperature of 97°. The pulse continued at 80, the respiration at 20, and there were no signs of meningeal involvement. The discharge from the right ear was very much less and was odorless. The patient died at 1 p. m., March 10th.

Death was caused by secondary thoracic complications of mastoiditis. I did not see the case from the time that the acute symptoms had subsided, until autopsy.


Temperature (Fahrenheit)
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98°
99°
100°
101°
102°
Pulse
Respiration
Day of Month
Hour
16 76
20 78
26 110
24 118
24 106
26 110
20 106
20 82
18 80
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20 84
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AUTOPSY. March 10th, performed by Dr. G. Y. Rusk. In the autopsy, the following conditions were disclosed:

1. Double purulent otitis media and mastoiditis. The mastoid processes were extensively involved with pus throughout the cells, and there was softening of the bone, including the antrum and the neighborhood of the tegmena. The mastoid pus showed streptococci in short chains. No perforation of the bony walls of either middle ear tract was seen. The left tympanic membrane was intact. The veins and sinuses were normal.

2. Acute pericarditis.

3. Pulmonary edema with diffuse purulent bronchitis. In the acute pericarditis and purulent bronchitis, the bacteriological infection was the same as that in the mastoiditis,—streptococci.

CHARTS.

SPECIMENS.

SUMMARY. This case if of unusual interest for the following reasons:

(1.) Double mastoiditis existed and resulted fatally without symptoms threatening a speedy fatal termination.

(2.) In the **right** ear, the only existing symptom of mastoiditis at the time of death was mild middle ear inflammation with a slight non-fetid discharge.

The mastoiditis passed the acute stage, the swelling, edema, mastoid tenderness, and pyrexia disappeared 13 days before death, but the mastoiditis persisted and resulted fatally.

This fact proves that mastoiditis may exist and result fatally without the classical symptoms. There was no pain or tenderness, no swelling, no edema, no rise of temperature, no vertigo or no nystagmus.

(3.) The **left** ear at no time gave any symptoms of mastoiditis with bone softening. This fact proves that mastoiditis may exist and result fatally with no classical symptoms, and without perforation of the tympanic membrane.

(4.) This case also proves that with the absence of symptoms of presence of mastoiditis, the diagnosis must be determined from the history of the case.

(5.) Mastoiditis may result fatally by causing metastatic complications in distant parts of the body.

(6.) Mastoiditis may result fatally without local symptoms.

(7.) There were no symptoms of toxemia.

(8.) There were no signs of meningeal involvement.

CONCLUSION. The history of this case offers a strong argument against our being misled by an apparent convalescence from mastoiditis. When bone involvement is first evident, an immediate mastoid operation is demanded.

Discussion.

Dr. Edward B. Dench, of New York city, asked Dr. Bryant whether he thought the second mastoiditis was secondary to the first, infection taking place through the blood.

Dr. Bryant replied that autopsy contraindicated this.

MENIERE'S DISEASE. REPORT OF A SERIES OF CASES OCCURRING IN ONE FAMILY.

By D. HAROLD WALKER, M. D., Boston, Mass.

In looking through recent literature on the subject of Ménière's disease, one meets a great many conflicting theories and facts as to just what Ménière's disease is, and whether or not the pathological changes described, are sufficient to cause the symptoms commonly called "Ménière's symptom-complex," consisting of nausea, vomiting, tinnitus, vertigo and deafness.

It is, with the hope of causing a discussion of the subject by the members of the society, that I report the following cases.

The family I shall describe is of unusual interest, not only because of the forms of the disease it illustrates, but also because of its having affected members of several generations of the same family and collateral branches. The relationship is shown by the accompanying chart. The grandfather of the oldest living generation had definite attacks of vertigo; whether there were the usual accompanying symptoms is not known. He died at the age of ninety-five years. The next generation is free, the attacks appearing in the third generation, and I will hastily describe the symptoms of those I was able to examine. First, Mrs. T., aged sixty-nine years. In perfectly good health, except for considerable tinnitus. Blood pressure 175 mm. Hearing good until the first attack which occurred fourteen years ago at the age of fifty-five. She was sitting at the table, when suddenly she experienced the most intense rotary vertigo and it seemed as if she were being thrown to the ceiling. Nausea, vomiting and tinnitus followed immediately. The attacks came as often as every other day and would last from a few hours to two days, and were always preceded

by tinnitus. At times she has been awakened out of a sound sleep by an attack, the bed rocking from side to side. Treatment had no effect and six years ago Dr. E. A. Crockett, of Boston, attempted to remove the stapes. The incudo-stapedial joint was cut, but the crura of the stapes broke in the attempted removal. This operation had no effect on the frequency or the severity of the attacks. Both drum membranes are slightly retracted and thickened and the left shows the healed incision of the operation. The hearing in the right ear is still good but almost totally gone in the operated ear. Since seeing her two weeks ago, she has had an attack, preceded by increasing tinnitus. The second case is a sister of the above, a Miss S. She is sixty-six years of age, always rather pale and anaemic, of slight build, just the opposite of Mrs. T., who is, as she expressed it, "full blooded." Blood pressure 130 mm. Hearing before attacks normal. The first attack appeared at the age of forty-one, and it would seem as if she were being thrown through the side of the house. She also, had rotary vertigo, nausea and vomiting tinnitus and slight deafness. She has had comparatively few of these severe attacks, but has frequent impressions of uncertainy of equilibrium. Both drum membranes show slight thickening and some retraction. The third case is that of a brother whom I did not examine. He is seventy-one years old and has had two severe attacks, the first sixteen years ago. Both of these attacks were sudden and accompanied with the usual symptoms. The hearing is still good. We now turn to another branch of the same family; here are two cases of the same generation. First, Mr. S., age seventy-two years. Apparently in the best of health with a blood pressure of 150 mm. The hearing in one ear totally absent since childhood, cause unknown, in the other ear, very good. His attacks are similar to the others, but not so frequent. They would last three and four days and during the time, he could not raise his head above the pillow. Both drum membranes are very clear with practically no retraction. Hearing in one ear present for all tones but the lowest. Mr. S. married his

second cousin. Mrs. S. is sixty-eight years of age and in good health with a blood pressure of 120 mm. There is a history of a blow on the back of the head in childhood. Her first attack came at the age of thirty-two, the hearing previous to that time was good. She had a sensation of falling from a great height and suffered great exhaustion. The attacks would last from three to five hours and recur as often as once a week. The tinnitus was of an intense metallic character. She has had no attacks for the past twenty years. The hearing in the right ear is gone, but all but the lowest tones heard in the other. Both drums show considerable retraction and thickening. Thirdly, the son of the last two cases has had attacks. He has been slightly deaf for twenty-five years. The first attack occurred in Boston twelve years ago, curiously enough he was able to ride a distance of five miles on a bicycle, but upon arriving home could not walk and fell to the ground. He had nausea, vomiting and tinnitus for three days. The hearing was markedly affected in both ears but not entirely gone until three years ago, when after an attack, he became almost totally deaf, only hearing a shout in one ear. A brother has had attacks of vertigo, but not associated with the other usual symptoms. A descendant of a third branch of the family has had a severe attack recently and a daughter of another brother of the fourth generation is ill at the present time. One ear has been deaf for several years and now the other one is affected, although I am told this last ear affected, is better. Besides the above cases there are two more who have died. They also had attacks of vertigo, I do not know how severe.

When one mentions vertigo, it suggests countless theories and causes and Barany, in his historical development of the general symptomatology of vertigo, speaks of it as having been known in the time of the ancient Greeks and Romans, and Walther, in a dissertation in 1727, gives a very clear picture of the now known "complex." He also speaks of ocular movements as being a cause. Hippocrates mentions vertigo as accompanying discharge from the ear. During

the last century, Itard in 1821, Flourens, Purkinje and Brown-Sequard began their experimental work upon animals, and proved the importance of the semi-circular canals in co-ordinating movements. But it was left to Ménière to associate the semi-circular canals with the symptom of vertigo in persons suffering from ear disease. Ménière was physician in the National Deaf and Dumb Institute in Paris. For years he had noticed vertiginous attacks in persons perfectly well, such attacks previously, and at that time were supposed to be due to cerebral complications. The patients, however, fully recovered and were as healthy after an attack as before. In 1861, he observed the case which made his name famous. A young girl who had become thoroughly wet by riding on the outside of the post coach, had a sudden attack of vertigo nausea, vomiting and tinnitus and became totally deaf, finally dying upon the fifth day. At the autopsy nothing was found except an exudate in the vestibule, the canals and cochlea being normal. As Barany states, the report was writen from memory, but one ear was examined and it was before the days of exact bacteriological and pathological methods, consequently open to question. Since then a great many cases of Ménière's disease have been reported, but the number which exhibit the true apoplectic form is probably small. Politzer, in the latest edition, was able to collect but seven authentic cases. Heerman of Kiel has collected a series of fifty cases and published the pathological reports of fifteen. The Ménière symptoms occurred in cases afflicted with a primary disease, such as syphilis, lukaemia, typhoid, Tabes tumor of the brain and injury from accident. The exudate found in the labyrinths of these cases is such as one would expect from a hemorrhage either recent or organized.

In making an analysis of the cases reported above, the question arises as to the cause of the attacks described; why should they occur in members of the same family and in collateral branches. Here is a long-lived, sturdy Puritan stock, having settled in X in 1639. There is no history of any constitutional disease, such as syphilis, etc., nor is there

a history of death due to heart or kidney disease, and but one death caused by apoplexy. With the exception of the last mentioned case, they are not a nervous or neurotic people, but a race living in the country and unusually strong mentally and physically. The blood pressures, except in one instance, are normal. From the severity of most of the attacks, the mild changes to be found in the middle ears, the fact that the hearing was good previous to the attacks, and which has been retained in a few of the cases, leads me to assume that we can exclude the middle ear as a cause. If the attacks were due to hemorrhage, it seems strange that so many members of the family should be affected and also that the hemorrhage should be confined to the ears, especially as circulatory changes are lacking in every case. Again, nearly every case reported in literature, where hemorrhage exudates have been found in the labyrinth, has been due to some primary cause, either constitutional disease or injury. In other words, the disease called Ménière's disease, seems to be nothing more nor less than labyrinthine hemorrhage and should be called by that name, and also the condition is a secondary process. It seems to me that the cause of the attacks described must be sought for in some anatomical anomaly which has been transmitted from generation to generation.

We assume that glaucoma is a disease of the eye characterized by an increase in the intraocular pressure. This condition is either due to a change in the aqueous fluid or a closure of the canal of Schlemm with a consequent increase in pressure because of insufficient outlet. The decrease in size of the canal of Schlemm is known to be inherited. In the labyrinth we have an analogous condition existing, with the addition of a compensating pressure organ. Let us speak in detail of a few anatomical structures. First there is the aquaeductus cochleae, which, according to Schwallbe is ten mm. long, and connects the scala tympani with the subarachnoid space. It contains lymph vessels and transmits a small vein from the cochlea. Why is this anatomical arrangement not analogous to the arrangement in the eye?

As will be remembered, the membranous vestibule occupies only a portion of the osseous vestibule, the space being filled with the perilymph. This fluid is renewed from the subarachnoid space. Is it not possible that the cases described have an abnormally small osseous aquaeductus cochleae, which is a distinctly inherited anomaly, and if that is probable it is not difficult to understand that an increase in the amount of the endolymph would exert a pressure in all directions on the perilymph and in the lymph spaces, thereby shutting off the venous outlet with resulting tinnitus and vertigo from congestion. If this pressure were marked a lasting injury to the hearing and a general disturbance of equilibrium would result. The same condition might result from an increased viscosity of either the perilymphor or endolymph and a consequent rise in pressure. But there is still another anatomical organ to be considered. Namely, the saccus endolymphaticus. The aquaeductus vestibuli contains the ductus endolymphaticus which is about seven mm. long and .25 mm. in diameter, and which connects the vestibule with the saccus endolymphacitus, a blind sac situated upon the posterior portion of the petrous bone and underneath the dura mater. I believe that this organ may act as a compensating chamber or an equalizing organ for maintaining a constant pressure in the vestibule. The sac is under the dura mater, is connected directly with the vestibule just where the least variation in pressure would be reflected with a resulting expansion of the sac and an equalizing of the pressure. When one realizes the minute movements of the labyrinth, it is not difficult to suppose that a very slight compression of the ductus endolymphaticus in an already narrowed osseous canal would either lessen or entirely defeat the physical functions of the saccus endolymphaticus and vertigo with or without deafness would result. These theories might explain tinnitus and a great many attacks of vertigo, especially when associated with stapes fixation and a thickening of the membrane of the round window. Also where there are sudden variations in arterial pressure as from vaso motor disturbances.

In conclusion, I would like to suggest that the name Ménière's disease be applied to such cases as described by Heerman, namely, those due to hemorrhage into the labyrinth, secondary to some constitutional or known cause. That the name auditory vertigo be applied to such cases as I have described, and to all other forms which occur in persons with known defects in the organ of hearing.

I wish to thank Dr. Allen Greenwood, of Boston, for his courtesy in enabling me to report these cases.

Discussion.

Dr. George E. Shambaugh, Chicago. The paper presented by Dr. Walker is of very great interest as it appears to emphasize a new fact in regard to the interesting subject of Ménière's disease; that is, that there may be marked hereditary predisposition for the development of this condition. The term Ménière's disease has been used too loosely in the past, often being used as synonymous with the term Ménière-symptom-complex. Our knowledge of the pathology of the labyrinth has increased very greatly since this first contribution by Ménière in 1864. We know now that there are a large number of conditions which will produce the symptoms pointed out by him as characteristic of the condition which has since been called Ménière's disease. Any acute condition involving the labyrinth of the ear of the eighth nerve will produce the symptoms. By the term Ménière's disease, however, we mean an acute hemorrhage into the labyrinth. It is not at all easy to make a differential diagnosis between some of the conditions which give rise to the Ménière-symptom-complex and the true Ménière's disease, i. e., hemorrhage in the labyrinth. There are certain general diseases, such as leucamia and pernicious enemia, where a sudden attack of the Ménière-symptom-complex, with persistence of the deafness, would seem to justify the conclusion that we have to deal with a hemorrhage in the labyrinth. There are, however, a large number of cases presenting the typical attacks of the Ménière-symp-

tom-complex which cannot be attributed to a hemorrhage. These have often been referred as cases of neuritis of the acoustic nerve. It is still a subject, however, about which there is a good deal of mystery.

Dr. Hiram Woods, of Baltimore, asked about the term "Ménière's symptom" as compared with "Ménière's disease." In this connection he referred to the case reported by Dr. Sheppard, where two or three capable men made the diagnosis of Ménière's disease, and yet relief came after operation for manifest middle ear trouble. Dr. Sheppard had explained it by saying it was caused by pressure; but would it not be necessary to modify this by saying where the pressure is? The speaker cited a case where attacks of vertigo would come without the slightest warning. The patient finally died of cerebral hemorrhage. Another case was that of a man who was totally deaf from middle ear sclerosis. He suffered from attacks of giddiness, often in the middle of the night. In the first case there was no evidence whatever of middle ear disease. The attacks of vertigo were diagnosed as Ménière's disease, and the patient died of cerebral hemorrhage. The other showed symptoms of middle ear disease. Are these cases the same?

Dr. Walker, in closing the discussion, called attention to the fact that he had distinctly said that the term should be confined to cases where there is hemorrhage. In other words, Ménière's disease is always a secondary disease. But where the symptoms are due to increased intra-labyrinthine pressure the condition should not be called Ménière's disease at all. The reason why relief from the attacks is secured by purging and by treating the nasopharynx, is, that in this way the intra-labyrinthine pressure is reduced.

ERYSIPELAS AS A COMPLICATION OF MASTOID DISEASE.

By GEO. L. RICHARDS, M. D., Fall River, Mass.

In 1907, Dr. C. R. Holmes, of Cincinnati, published a most elaborate paper on the etiology of erysipelas and its relation to the nasal cavities. This paper was based on a comprehensive study of the literature of the infectious organisms present in the upper air tract, supplemented by a large number of clinical reports, both published and hitherto unpublished, showing the presumptive relation of facial erysipelas to infectious diseases of that portion of the mucous membrane of the upper air tract which has continuity of surface; namely, the nasal passages with their accessory cavities, Eustachian tubes, mastoid cells, the fronto-nasal ducts and conjunctivae. These studies show that streptococci are nearly always present somewhere in this region, their pathogenicity varying considerably, in some cases largely losing their naturally virulent character, but probably becoming more virulent when planted on more suitable soil. In other words, the individual becomes practically immune to them at the site of their original location, but not immune to the same streptococcus when transplanted to another place, and when the resisting power of the individual is for any cause reduced.

Dr. Holmes recorded one hundred and two cases of facial erysipelas occurring in connection with disease of the nose and accessory sinuses or following upon acute and chronic mastoid disease. These cases occurred in the practice of sixty-nine American surgeons. They do not include those cases of the same character gathered from the literature. His conclusions were, that the identity of the streptococcus as the microbal cause of erysipelas seems now to be a definitely established fact. This streptococcus is of slow

growth and of variable morphology and pathology whenever its environment is changed. It is probably but rarely air-borne, requiring direct personal contact for its transference.

The streptococcus is of nearly constant presence in the normal nose and neighboring spaces. It may lie latent for months or years without betraying its presence, but this quiet existence may be broken at any moment by some temporary or unknown cause inducing virulence in the micro-organism through some unfavorable condition supplying the environment necessary to incite the organism into virulence, with the attendant formation of toxins. The extreme frequency of facial erysipelas has been noted for many generations, and Dr. Holmes' studies showed an apparently causative relationship between facial erysipelas and suppuration in the regions studied. Even in those cases in which no bacteriological examination was made, there was sufficient presumptive evidence to assume that the erysipelas-producing streptococcus was probably present in the discharges of the disease which was under treatment at the time of the appearance of the erysipelas. These cases of erysipelas occurred in the hands of careful surgeons, were not due to neglect, occurred in hospitals some of which were new, in others where no cases of erysipelas had occurred for many months, and unless some such explanation as this satisfies, the cause must be regarded as unknown.

In most of the cases reported from the statistics gathered by Dr. Holmes the erysipelas does not seem to have had any serious effect upon the progress of the original disease. There were twelve deaths, but only in five could it be said in fairness that the erysipelas was the cause of the fatal result.

Observations of substantially the same character as those of Dr. Holmes are also made by Dr. Welty in an article on "Acute and Chronic Suppuration of the Ear and Nose as the Direct Cause of Facial Erysipelas," presented before

the Section on Laryngology and Otology of the American Medical Association in June, 1906.

In addition Welty thinks that epidemics of erysipelas are nothing more than direct wound infection carried by surgeons or nurses, and that the so-called idiophatic erysipelas is a misnomer. The fact that erysipelas does not occur in every case of streptococcus infection is due to the character of the streptococcus and the immunity of the patient. Assuming these observations to be correct, the practical conclusions would seem to be:

First, the occurrence of a case of facial erysipelas in a hospital ward is not of necessity any reflection on the surgeon or the hospital, as it arises in nearly every primary case as an auto-infection.

Second, erysipelas is probably much less contagious by direct contact than has been supposed and should therefore be less feared, nor is there much likelihood of an epidemic of erysipelas occurring from a single case of erysipelas, provided the infection is not transmitted by the attendants, instruments, dishes, etc.

Third, as a routine measure in all operations on the mucous membrane of the upper air tract it would be advisable before operation to cleanse the nose as carefully as possible, though it is by no means asserted that thereby every streptococcus or pneumococcus germ can be destroyed.

The three cases now to be described I offer as a contribution to the clinical history of facial erysipelas and as substantiating the foregoing conclusions. This point of view of the general etiology of erysipelas does not seem to have been as yet sufficiently grasped by the general surgical profession. In a very recent article on erysipelas in a general medical encyclopedia I find no mention of this theory of the causation of facial erysipelas.

Case of M. J. D., 55 years of age, though looking much older, slight tendency to alcoholism; otherwise general health good. Seen first April 5th, 1908, complaining of otitis media; next day incised the drum; following day, ear discharging freely, some mastoid tenderness; noticed for

first time erysipelatous blush at the outer angle of the left nostril. This slowly involved the entire left side of the face, reaching the region of the mastoid last of all, extending from this point backward to the mastoid and the corresponding side of the neck, temperature going to 104½ and becoming normal on April 15. The erysipelas lasted about 10 days, the blush disappearing in about a week. By April 29th the erysipelas had completely cleared up, the ear was discharging, there was considerable loss of tissue of the membrana tympani. During all this time there had been mastoid tenderness. On May 7th a thorough operation for acute mastoiditis was done. The mastoid was very large and very cellular, and there was purulent debris in every cell. Recovery slowly took place, the last filling-in not being completed until June, owing to the presence of a small bit of carious bone tissue corresponding to the roof of the antrum cavity. This was finally curetted away, uncovering the dura for a short space, after which the cavity promptly filled in.

Now as to the relation of cause and effect. At the time of the advent of the erysipelas no one connected with the case had had anything to do with a case of erysipelas for months. The patient was at his own home, so there could be no hospital infection. It seems to me probable that the patient with his own fingers carried the infection from the ear to the corner of the nose, probably with the finger nail. This is the more likely as he was inclined to feel around his ear with his fingers, and at first the ear was only wicked and not bandaged. I do not think the erysipelas caused the mastoid, as I was suspicious of beginning mastoid before the erysipelas began. The patient and his family physician were both loth to have the mastoid operation done, and he was out and about and came to my office after the subsidence of the erysipelas, the discharge and the mastoid tenderness continuing until the operation was finally done. The erysipelas was due to direct infection from the purulent discharge of the middle ear.

No bacteriological examination of the discharge was

made, but on account of the extensive destruction of the mastoid and the subsequent slow recovery, the presumption is all in favor of the streptococcus infection.

Case No. 2, T. P. S., a dentist, aged 33, was operated on at St. Anne's Hospital for an acute mastoid on February 23d, 1908. The operation had been delayed, owing to failure of the patient to give his consent, and the mastoid, which was large, was very much broken down, every cell in every direction being involved. After he had begun to leave the hospital and come to my office for treatment, but while the posterior wound, especially in the attic region, was still open, he developed about April 1st a facial erysipelas, invading the entire left side of the face, front and back, and going back nearly to the vertex. It ran the usual course of an erysipelas, seeming in no way to interfere with the healing process of the mastoid. There was no other case of erysipelas in the hospital, nor was there any reason to suspect that either the surgeon or any attendant had carried the disease to the patient. Unfortunately, no bacteriological examination of the ear secretions were made, but from the intense degree of infection and tedious convalescence, it is probable that the case was one of streptococcus infection from the ear secretions. Recovery from the erysipelas took place after the usual time. The mastoid wound healed perfectly and the accompanying facial paralysis almost completely disappeared.

Case No. 3, Captain H., aged 65, retired officer of the United States revenue cutter service, had suffered for many years from an empyema of the right antrum. On January 22, 1910, an operation was done through the naso-antral wall, a portion of which was cut away so as to give permanent drainage. The after treatment of this was uneventful for the first few days, and the patient was up and about, coming to the office for treatment. On January 31st, nine days later, he developed facial erysipelas in the same side from an abrasion at the angle of the nose. The erysipelas was at first on this one side, then involved the whole face. This ran the usual course and in the convalescent stage he

seemed to acquire a general streptococcus infection and was quite sick for a number of days. No case of facial erysipelas had been seen recently, either by his family phyiscian or myself. The amount of pus in the antrum was small, but there is no doubt that the source of infection was in all probability from the nasal or antral secretions.

A coincidence, and perhaps in the nature of cause and effect, though I am by no means certain of it, is the fact that my office assistant, who cleaned up the instruments after an examination of the nose at the time that this case of erysipelas was in the early active stage, herself developed erysipelas of the face nine days later. The physician who attended her, however, did not believe that she got the infection from this source, as he had under treatment two or three cases of sporadic erysipelas at the same time. She left work, complaining of fever and rheumatism, the erysipelas developing two or three days later. The disease ran the usual course and she was not very ill.

These cases are offered as a contribution to the subject of the etiology of facial erysipelas. The possibility of its occurrence after operations or infection of the upper air tract and adjacent communicating regions is to be borne in mind, though its occurrence under such conditions may be unavoidable.

Discussion.

Dr. Wendell C. Phillips, of New York City, called attention to the fact that cases of post-operative eryipelas are now taken care of in the Manhattan Eye, Ear and Throat Hospital instead of being removed to other hospitals. As a rule such patients are sent to other hospitals to be treated for the eryipelas, and when they return to the special hospital for treatment of the mastoid wound it is found to be in a horrible condition. He considered it the duty of the hospital receiving and operating upon the mastoid cases to treat them for whatever conditions may arise.

Dr. B. Alexander Randall, of Philadelphia, emphasized two points brought out in the discussion: first, that there may be some local immunity, so that there is less danger of carrying the germs of erysipelas to the neighboring tissues than to some more distant field; and second, that while the care and isolation of the patient is important, care on the part of doctors and especially the nurses to prevent spreading the infection, is more important.

Dr. Walter B. Johnson, of Paterson, N. J., thought that while it is important that operative cases be kept under observation, it is a question whether one is justified in exposing other operative cases to erysipelas. If a hospital retains such cases it should have facilities for isolating them. Inasmuch as it is not known how the streptococci of erysipelas pass from one person to another, and it is impossible to trace the course of infection from one to another, such patients should be very carefully isolated if they are to be kept under observation. A considerable epidemic of erysipelas had recently occurred in the Paterson General Hospital and the sanitary committee undertook to prevent the occurrence of other cases, but they did occur. The cases with the disease were immediately removed from the hospital, the rooms were immediately washed down, the beds repainted and everything thoroughly cleaned, and still the disease progressed. Unless erysipelas patients can be removed to a place where new operative cases are not in danger of infection, he did not think they should be kept in the institution, even though they lose the skilful attention which the mastoid or other wounds should receive. Some surgeons absolutely refuse to operate on other cases until the erysipelas cases are out of the hospital and the rooms properly disinfected. Until it is definitely known what the erysipelas germs are and how they differ from other streptococci, and until a certain method of preventing infection is discovered great caution must be exercised, no patient with an open wound should be exposed, and no new operation

should be performed unless the operator was absolutely sure there was no danger of infection.

Dr. George A. Leland, of Boston, said, in his experience erysipelas affects a patient in one bed and not any other patient in the near vicinity. He lately went through the ward where these cases are segregated in the Boston City Hospital and asked the patients if they had any irritation in the nose; every one had cracks in the introitus. When these fissures were healed, in a good number of cases, both private and hospital, the tendency to erysipelas disappeared. He had never seen any real practical reason for isolating these cases. He cited a case of supposed mastoiditis complicating erysipelas in which he was called upon to operate. He made the diagnosis that the mastoid was not involved, but out of deference to the opinion of the eminent surgical consultant, he allowed the aural house surgeon to do the ordinary mastoid operation right through the thickened erysipelatous skin. The mastoid was found to be perfectly healthy. Healing took place as promptly and as completely as it would have done through the normal skin, and the erysipelas remained external; and the patient kindly and promptly recovered. Since that time the speaker has been less afraid of erysipelatous infection being transmitted to other patients, or to other wounds.

Dr. Richards, in closing the discussion, agreed with Dr. Phillips and Dr. Leland concerning the danger of infection. He could not recall an instance of infection by direct contact of one patient with another in twenty years. The morphology of the streptococcus of erysipelas is very remarkable, the germ sometimes seeming to be so, inactive in one locality and active when transferred to another. While undoubtedly direct contagion may and does occur, erysipelas is not so contagious as was previously supposed.

*A FURTHER CLINICAL STUDY OF THE VASCULAR THEORY OF TINNITUS AURIUM.

By JESSE WRIGHT DOWNEY, Jr., M. D., Baltimore, Md.

At the suggestion of Dr. H. O. Reik, and with his valuable guidance and advice, it has been my privilege to undertake in his clinics at the Johns Hopkins Hospital and at the Baltimore Eye, Ear and Throat Hospital a further investigation from a clinical standpoint of the vascular theory of tinnitus aurium. The foundation for such observation is based entirely upon two papers presented before the American Otological Society in 1902-'03 and published in the Transactions of the Society for those years.

In these monographs Dr. Reik has given the results of a series of physiological laboratory experiments undertaken by him to determine the cardiac and vascular effects of irritation of the tympanic membrane and middle ear and of increased intratympanic pressure and it may be well to review and briefly quote his findings and conclusions. In his first paper, The Cardiac and Vascular Effects of Operations Upon the Middle Ear, he has shown that irritation of the sensory nerve supply of the tympanic membrane and middle-ear is followed by a vaso-motor depression evidenced by a fall in general blood pressure combined usually with a cardio-inhibitory action. Such results tend to show that the faintness and sense of depression so often complained of by patients undergoing some trivial or painless middle ear procedure are due, in all likelihood, to a true temporary vascular hypotension, a much more plausible explanation than that offered by the theory of increased intralabyrinthine pressure. Physiologically the experiments were most interesting for, as the author has noted, they presented an exception to a general functional rule in view of the fact that sensory nerve stimulation is almost invariably followed

*Read by title.

by a rise in arterial pressure whereas in the situation under discussion vascular tone was diminished.

For the further study of this phenomena a second series of experiments were commenced with the purpose of studying the effect of increased tympanic pressure. Such a condition was found to have the same influence upon the circulation as had had mechanical irritation, viz., a lowering in blood pressure. "It is not necessary," the writer tells us, "that the tympanum should be filled with fluid to produce a rise in intratympanic pressure and a fall in blood pressure from a vaso-dilator effect. The presence of any exudate in the tympanum can be accounted for only by supposing the previous existence of inflammation and as swelling of the tissues is a constant accompaniment of inflammation it is reasonable to presume that the swelling may, of itself, cause sufficient irritation to bring about some depression." Having conclusive evidence that the calibre of the vessels was effected by experimental methods simulating those pathological conditions usually attended by tinnitus, the natural application of such proof was to substantiate a hypothesis advanced by Dr. Theobald, "that tinnitus aurium in almost all cases might be attributed to the existence of vibrations excited in the walls of the blood vessels of the labyrinth by the friction attending the circulation of the blood, which are capable of imparting to the labyrinthine fluid and thence to the terminal filiaments of the auditory nerve, impulses capable of giving rise to the sensation of sound."

The proof of the vascular theory may be said therefore to rest upon the following points:

1st. That the auditory nerve is capable of perceiving and translating into sound impulses due to a change in the blood current.

2nd. That a local change in vessel calibre and blood current is certainly present in those pathological middle ear conditions attended by tinnitus and perhaps a general vasomotor reflex is also occasioned as the result of stimulation of the sensory nerve supply of the tympanum.

3rd. That this change in calibre is a dilatation and the reflex a vaso-motor depression.

It is not my purpose to go into the details of the theory itself, this has been completely and ably done by Dr. Reik in the third paper of his contributions to the subject.* In it he has reviewed thoroughly the weak points in the generally accepted explanation of tinnitus and given convincing deductions concerning the probability of vibrations in the walls of the blood vessels being capable of stimulating the auditory sufficiently to produce what we know as sound. That a variation in calibre of the vessels is a cardinal part of all inflammatory conditions is an unquestionable axiom, that this variation is a dilatation and that the same loss of tone may be produced by stimulation of the sensory nerve supply of the ear to such a degree as to produce a reduction in general blood pressure has been demonstrated by laboratory experimentation. Assuming therefore that tinnitus aurium is due to a change of calibre of the blood vessels of the labyrinth such a change being specifically a dilatation permitting an increased volume of blood to pass through and causing a radiation of sound waves which, though small in amplitude and intensity, are so magnified by the proximity of the vessel walls to the terminal filiaments of the auditory nerve that they become appreciable, what clinical evidence may be offered to support the belief. In those individuals suffering with tinnitus is there evidence of present or past tympanic irritation sufficient to have caused a disarrangement of vascular tone? Has this irritation been sufficient to cause not only an anomalous hypotension in the vessels of the labyrinth but a general low tension as well? The first question may easily be answered in the affirmative as will be shown later, the second it seems probable must stand open to surmise on account of many factors more or less hypothetical. Reik has cited ten cases in which he found a lower systolic pressure than the standard average blood pressure for the given age. His measurements were

* A Consideration of the Vascular Theory of Tinnitus Aurium. H. O. Reik.

made along a plan described by Thayer and his readings compared with a table of normal pressures by decades compiled by the same author.*

"I would not want to be understood as drawing any positive conclusions from this small number of cases," he says, "further investigation along the same line will demonstrate in time whether these cases are truly representative of the whole class." As a preface to my report I have already given as its object the continuation of this investigation.

In the beginning there seemed but one point of interest, viz., have persons with tinnitus a low blood pressure? To measure blood pressure is without difficulty, to apply arbitrarily the results of such measurements to prove the point at issue has been found to be impossible and in presenting the first fifty some cases of the series the idea it is not to draw any positive inferences yet a while but to offer, as a preliminary to a continued research, such evidence as has been obtained pro or con.

Before entering into the clinical findings of the cases examined I must ask your attention concerning certain facts and factors which have been found both a help and a hindrance in accomplishing accurate observations. The cardio-vascular theory of tinnitus aurium does not make the condition an entity in itself, it offers only, an explanation for a symptom often the most distressing of the pathological state of which it is a part; consequently a study of this one symptom must take into account all the other changes with which it is connected and which as a whole make up the vicious circle. We assume that the aural sounds are due to the perception of variations in the blood current, such variations being in turn due to irritation within the middle ear or elsewhere. The first part of the postulate must remain without definite proof, the existence of an etiological element capable of producing vaso-dilatation can be easily verified by considering the pathology of the condition of which the tinnitus is a symptom. With this latter in view

* Amer. Jour. Med. Science, March, 1904, Vol. CXXVII., No. 3.

as thorough aural examination as time would permit was made in each case to be reported and according to the condition found they were divided into the usual classes of:

(1) The canal affections.	(a) Acute Ex. Ot. Media.
(2) The middle ear affections.	(b) Subacute Ex. Ot. Media.
(3) Affections of the labyrinth.	(c) Chronic Ex. Ot. Media.
	(d) Otosclerosis.

Hearing tests were made to confirm the seat of the trouble, for lateralization when one ear alone was declared at fault and to estimate the degree of the deafness. The appearance of the drumhead was the chief guide in determining the presence of existing or past trouble, and when found, the possible cause of the abnormal state of the ear, e. g., hypertrophied turbinates and degenerate tonsils were taken into account.

It needs but a passing thought in respect to the morbid anatomy of the affections of the middle ear to realize that the vessels must undergo dilatation; it may not be amiss to say at this time that it is much easier to infer that a local vascular disarrangement is not only occasioned but remains more or less a permanent result of an inflammatory process whereas the general vascular depression from the sensory nerve stimulation is either a transient reflex as shown in the experiments of Reik or its range limit so influenced by the many factors outside the ear that the measurements can never be dogmatically accepted as due to a referred aural irritation.

The local examination of the ear being completed the question arose of estimating the general systolic blood pressure; in view of the more recent works along this line it was found that the method employed and particularly the standard accepted by Dr. Reik could not be utilized in such a positive manner as seemed possible at the time of his work.

Blood pressure is a variable phenomena not only in different people but in the same individual under the same conditions of general health; it varies with the age, the habits,

the general health, the vocation and the sex in a number of persons examined and is influenced in each individual by many factors and reflexes, varying with the time of day, the posture, the amount of exercise taken, the emotional state, etc.; there is a standard above which or below which we may consider abnormal, but the range allowed is wide, so wide that the upper and lower limits are of more value than are averages and the differences between individuals so variable that it is impossible to use one as a standard for another. In considering the subject in connection with tinnitus aurium we are dealing with a theory that accredits low systolic pressure if found to be due to a sensory nerve irritation within the ear. The question may be asked is this a necessary reasoning and would not a low pressure from any cause be accompanied by tinnitus? Theoretically I think the latter may be answered in the affirmative as has been already done by Reik when he mentions tinnitus as a concomitant of systemic condition; such as syncope or during convalescence from some affection marked by depression and a relaxed vascular system; this, however, lends weight only to the belief that the 8th nerve is capable of recognizing the change in blood current, there being no morbid process present in the ear itself. If we however are to accept the theory that tinnitus is due to low systolic tension occurring with no cause for a general vascular relaxation and as a symptom of further ear trouble, the ear must be held accountable for the low blood pressure, on the ground that laboratory experiments have proven that the nerve supply of the ear is capable of producing, when stimulated, just such an action.

Why such a reflex should be produced from this location and as an exception to a general law has not been explained. The maintenance of blood pressure depends upon four factors as follows:

1st. The energy of the heart.
2nd. The peripheral resistance.
3rd. The elasticity of the vessel walls.
4th. The volume of the circulating blood.

That some of these may be influenced by a reflex originating in the ear seems probable in view of the statement of Janeway in his text-book on blood pressure that "vaso-motor tone both general and local is markedly affected by reflexes of remote origin. They may be produced by the stimulation of any afferent nerve. The only such nerve in the entire body stimulation of which invariably lowers blood pressure is the 'depressor nerve.'" "The course of the 'depressor nerve' is through the 'vagus'" (Howell) and according to Janeway "the fall of blood pressure which follows strong stimulation of the vagi peripherally is produced by both slowing the heart and decreasing the systolic output." What effect the aural branch of the 10th might have in this connection and in connection with the subject under discussion I feel a hesitancy in saying, but that it has branches of communication with some of the nerves making up the complicated tympanic supply is undoubted and that it is capable of producing from this location a general reflex we have an example in the common experience of producing a cough from the introduction of an aural speculum too far into the external auditory canal. The remaining factors sustaining blood pressure may be passed over briefly with the statement that vaso-motor tone in particularly vascular areas varies constantly in response to local needs and from the pathology of the ear conditions interference, at least locally, can be proved to exist.

In offering clinical evidence, however, that low blood pressure is present and is influenced by the ear condition we are confronted by many difficulties, first of all as it has been said we cannot use one person as a standard for another even for an approximate comparison. We cannot, for example, say that a certain person has tinnitus because he has a tension 10 or 15 mm. Hg. lower than some other individual not so affected so long as both measurements come within the normal range; comparison can only be of value when the pressure found is below the range limit or when in the same person a variation is accompanied by a change in the symptom we are studying. If the blood pres-

sure is at a certain point today and the tinnitus is most annoying will there be relief from the noises tomorrow if the blood pressure is found to have a higher reading? A single estimation of the blood pressure in a patient suffering with tinnitus can prove nothing regarding the relation between the two, to be of any service we must have repeated readings in the same case.

I wish to present the findings in a series of fifty-two cases examined; the patients were not selected, but were taken as they came, the statement from them, "I have noises in my ears," being the guide to include them in the list. The usual aural examination was first made and the signs and symptoms noted and as a last step the blood pressure was measured. All the examinations were made in the afternoon between two and five o'clock, the patients as a rule came into the Dispensary from their work or had walked from home to the hospital. The blood pressure was taken with the patient in a sitting position, the cuff being used over the bare arm, though in some instances it had to be fitted over the sleeve; I have mentioned these particulars because each in itself is an element tending to cause a higher vascular tension than would be present could the estimations have been made in the ideal way of ascertaining pressure. The Cook modification of the Riva-Rocci apparatus was used with an *eight cm* armlet.

In beginning the study of these cases it was thought that a comparison might be made with the table of Dr. Thayer or some similar list of averages; besides the wide diversity of averages normally existing, Thayer's examinations were made with the narrow cuff, a method which gave readings generally much too high, in reaching any conclusions therefore each case had to be followed upon its own findings in repeated examinations and whereas this was possible in many it was not feasible in all.

In regard to the normal range of the systolic blood pressure Janeway gives in his text book 80 to 160 mm. of Hg. as the widest range consistent with health, but considers 90 to 145 mm. of Hg. nearer the absolute value.

His opinion on the point in which we are interested, expressed by letter, is as follows: "So far as the general limits of normal blood pressure go I should consider 110 to 145 normal for healthy male adults and about 10 mm. lower for women. The normal blood pressure is sufficiently variable so that the upper and lower limits rather than averages are valuable. I believe that the differences between individuals are probably greater than the differences in any particular individual due to such depressive effects as you are studying. For that reason deductions from your observations would be somewhat difficult unless you can find a patient free from ear disease and have consecutive observations until he develops the ear disease." Such observations as Dr. Janeway advises would of course be difficult to make, as it could hardly be possible to select a person who later is to develop tinnitus, and so the best way and the only one is to follow the course of those unfortunate enough to have already acquired the annoying condition.

The cases examined may be classified as follows: Males 18, females 34, the youngest being 18 years of age, the oldest 71.

15 were between 18 and 39 years of age.
18 were between 30 and 40 years of age.
9 were between 40 and 50 years of age.
8 were between 50 and 60 years of age.
1 was between 60 and 70 years of age.
1 was over 70 years of age.

36 had tinnitus in both ears, the noise being more marked in the right ear in two of these cases, in the left in eight. Thirteen had tinnitus in the right ear alone. The duration of the trouble varied from one week to five years, many were doubtful as to the length of time. Thirty-nine of the patients were in good health so far as they knew or so far as could be determined by a cursory examination. The following general diseases were noted:

Insanity	1
Valvular heart lesion in	1
Pulmonary tuberculosis	2
Nephritis	2
Insanity	1
Myocarditis	1
Neurasthenia	1

One patient was under a rigid gynaecological examination, two were under examination for nervous conditions and two were chronic alcoholics.

The ear examination showed the canal to be at fault in four, the middle ear in forty-two, the labyrinth in three. Three cases were examined in which no ear lesion could be found.

THE CANAL CASES.

The canal cases were Nos. 16-21-22-23. All four were found to have the canal plugged with cerumen, which was removed by syringing; before this was done, however, the blood pressure was taken. In three of the cases the blood pressure was found well within the normal range, in the fourth the pressure was 100 mm., a rather low tension which could be accounted for by the fact that the patient had been under treatment for pulmonary tuberculosis, from which she had apparently recovered. The interesting point in these cases is the fact that the syringing caused a decided drop in the blood pressure, measurements being taken during the irrigation and immediately after. Patient 16 fainted during the syringing, her pressure dropping to 80 or below and coming back gradually to the first index as she regained consciousness. In patient 23, who did not complain of tinnitus but of pain and in whom the test was made to determine the effect of the syringing, the pressure stayed at 100 mm., but fifteen minutes later the mercury registered 115 mm. All the cases were permanently relieved and the test gave conclusive evidence regarding the very first hypothesis advanced by Dr. Reik that the ill effects sometimes occasioned by such simple procedures were due to vaso-motor

depression and not to intra-labyrinthine pressure. The removal of an irritant in case 23 brought the vaso-motor tone up to a good normal value.(?)

THE LABYRINTH CASES.

Cases Nos. 35, 36, 50. There was no clinical evidence of increased labyrinth pressure from ossicular misplacement in any of these cases. One patient showed a normal blood pressure viz., 137 mm., the tinnitus and slight deafness had followed a loud explosion occurring near her during a 4th of July celebration. Her blood pressure was certainly not a high normal in view of the fact that she was a woman of 65, and one who had not to all appearances spent a life of ease.

The two remaining cases (36 and 50) had complications increasing the blood pressure, viz.—myocarditis and nephritis. Case 36 was a woman of 59 with Ménière's symptoms the vertigo being most marked, hearing negative and tinnitus slight; her blood pressure was 175 mm., she had a myocarditis with rather marked arteriosclerosis. Her high pressure was rather confirmatory of the hemorrhagic theory accounting for the train of symptoms simulating Ménière's Disease.

Case 50 was a young man of 22 who had been deaf since childhood, following a fever the nature of which he did not know; the hearing was nearly entirely lost. His blood pressure was 140 mm., due to a nephritic condition for which he was under treatment.

NO EAR TROUBLE.

Cases 12, 19, 49. Case 12 was a man of 20 who said he had been listless and weak for eight months; he came to the Hopkins for a thorough physical examination and was admitted to one of the wards where he was put through the usual rigid examination and no organic disease could be found. His tinnitus was not particularly annoying, just a low "hissing;" his drum-heads were normal, as was his hearing and likewise the blood pressure, viz., 115 mm.

Case 19 had been examined in several of the departments

of the Hopkins Dispensary and had been pronounced a neurasthenic. He complained of slight tinnitus in the left ear; drum-head and hearing normal. The man was an ex-prize-fighter and had an immense arm, his blood pressure was 145 mm.

Case 49 was sent from the gynaecological ward, where she had undergone an operation; she complained of occipital headache and slight tinnitus. Ears normal, blood pressure normal, viz., 145 mm.

In none of these cases was the tinnitus a prominent symptom and there was a "mental" element present in all.

THE MIDDLE EAR CASES.

The middle ear cases, as may be imagined, form by far the largest group of the series, the chronic exudative cases being the most numerous. While it is not possible to divide these patients into clear cut groups or to go into the details of each history regarding the relationship between the tinnitus and the blood pressure, some data was obtained which seems worthy of consideration. It may be said in respect to the blood pressure that the majority came within the normal range and as was expected more was gained from the study of the variations in an individual than from a comparison with what might be considered a standard. The effect of treatment, when successful or otherwise, offered some help in determining the relationship existing between the symptom and the supposed cause. The action of blood pressure raising drugs, notably Suprarenalin Extract, as advised by Reik, were watched in both their general effects and that upon the tinnitus with the idea of proving by this means the connection between the two. The usual methods of direct aural treatment were followed in all and when the restoration, so far as possible, of the normal tympanic state was obtainable, the effect upon the blood pressure was also noted.

As a matter of convenience the thirty-three cases have been divided into two classes; those in whom the blood pressure was found between 100 mm. or less and 127 mm. of Hg.

and a second class with the arterial tension above 127 mm. of Hg. In making this division it must not be supposed that either represent a criterion one way or the other, for in each will be found cases representing a high or low range dependent upon the sex, age and the general condition of the patient examined. In the first group (see Chart I) there were 15 cases, of this number eight had an arterial tension consistent with their age and sex, the remaining seven had pressures which, considering that the youngest was 40 and the oldest 71 years of age, and the highest pressure recorded 127 mm. of Hg. and that in a man of 71, may be considered rather low.

The so-called normal cases (3, 4, 5, 8, 9, 16, 33, 51,) showed the following points: Six were relieved by treatment and in each instance there was a corresponding rise in blood pressure. The relief seemed in direct relationship to the vascular tension, for as in patient 33 there was a recurrence of the tinnitus when the blood pressure became lower. All the cases showed definite signs of ear trouble. In the remaining cases of this class, Nos. 15, 32, 34, 42, 46, 44, 55, have been designated as having an index approaching hypotension. Case 15 was relieved when, under suprarenalin, her pressure rose 10 mm., when it again fell her trouble recurred and continued to be annoying regardless of treatment. Case 32 got transient relief from the use of suprarenalin. Case 42 is interesting from the fact that he complained of a hissing sound synchronous with his heart beat; he was under rigid examination in the ward of the Hopkins and was a highly nervous individual with many complaints the cause of which could not be determined. Daily blood pressure records showed his range to be as low as 80 mm. and never above 110 mm. Case 44, a man 71 years of age, with a tension of 115 mm., had some relief from the use of the Eustachian catheter and the bromides. No permanent benefit was obtainable in any of these cases or even any period of marked remission in the tinnitus; as in the preceding group, all showed more or less marked ear trouble; perhaps more than in the preceding cases were the tym-

panic changes well defined, the number however is far too small to allow such a statement to be positive.

In the second division of the catarrhal cases, viz.: those having a pressure above 127 mm. of Hg., two gave readings higher than could be considered normal, thirteen had tensions within the range and three showed a lower reading than one would expect in people of their age. The two cases having a high blood pressure were females; one (24) a woman of 34, with a tension of 165 mm., complained not of tinnitus but of pain, she gave a history of suppurative otitis years ago and there was a distinct scar in the posterior part of the drum-head and the malleous handle was adherent to the inner tympanic wall. She had had nephritis for eight years thus accounting for the high index found with the B. P. apparatus. Case 39, age 43, had a tension of 190 mm. of Hg. A specimen of urine was asked for but unfortunately she did not return to the dispensary and could not be traced.

The thirteen cases in whom the tension was normal are as follows: Five (2, 6, 13, 18, 20), received definite relief under treatment, in three of these (6, 20, 18), such relief was attended by a rise in the blood pressure. In two, suprarenalin extract seemed to have a favorable effect. Catheterization and massage were used in all. One patient, a woman of 41, with a B. P. of 150, was made worse by the use of the suprarenalin extract; another of the patients, No. 18, showed a definite relationship between the rise and fall in the blood pressure and the decrease and increase of her tinnitus.

Case 13 was seen during an acute tubal congestion, his drum-heads gave unmistakable evidence of a chronic exudative otitis of a long duration. His pressure was normal and his tinnitus was quickly cured by the use of the catheter. Cases 1 and 47 were unrelieved by treatment. Case 1 was a typical neurasthenic, suprarenalin hydrochloride was used by hypodermic injection, and although a fleeting rise in tension was thus produced, there was no let up in the subjective noises. Cases 7 and 10 obtained transient improve-

ment, suprarenalin had no effect on one and the use of the catheter gave the best results in the other, in whom there was but slight middle ear trouble.

Of the patients in whom the sphygmomanometers' readings seemed rather below normal, one or two things proved of interest. No. 17, a woman of 39, with a tension of 130, felt slightly better after the use of suprarenalin, although her blood pressure measured the same; later, after continued use of the drug and on a day when she had worked very hard, she was entirely free of her tinnitus and her blood pressure was at that time 140 mm. of Hg.

Cases (25) and (48), women of 53 and 50 years respectively, the latter having a secondary labyrinthine involvement, were both alcoholics. One was in a highly nervous state, complaining of hearing voices speaking to her; by restricting all stimulants she improved mentally, but her tinnitus still persisted as it also did in the other patient. Before leaving this group two cases must be taken alone; in one (28), an acute exudative otitis of one ear was accompanied by severe tinnitus; following a paracentesis the discharge became purulent. His blood pressure was normal and the tinnitus disappeared with successful treatment of the otitis. The other patient (41), complained of extreme tinnitus in the right ear, the membrane flacida was found thickened and congested and the canal irritated from some "home remedy" that had been used. Her B. P. was normal and the relief of the tinnitus was complete when the canal and middle ear had been restored to a normal condition.

These latter cases indicate, I think, the effect of local dilatation rather than a general vaso-motor depression.

Considered as a whole the exudative cases seemed to indicate a relationship between arterial tension and tinnitus, though the evidence offered is by no means conclusive. The treatment was most successful in those with a normal blood pressure, a definite rise in tension was noted when there was an abeyance in the tinnitus and, in several of the cases, a lowering of the tension accompanied recurrence of the

ear noises. The most persistent cases were those in whom the tension approached a subnormal index, these cases also exhibited a greater amount of middle ear trouble as indicated by the appearance of the tympanic membrane or by the degree of deafness present. In regard to the treatment one thing needs to be said about the use of blood pressure raising drugs; in the paper of Reik I have so freely quoted,* the question of the use of such agents has been discussed, he had had more or less success from the use of suprarenalin extract and considered it the safest drug having the specific action desired, its use there was continued in this series, the extract being used by mouth and in some few cases the synthetic product of the hydrochloride by hypodermic injection; by the latter method its effect was so fleeting, the tension returning in five or ten minutes to the original measurement, that its use by such means was abandoned. The action by mouth, as might be expected, was found to be uncertain, but in some instances it undoubtedly gave good results and in such an actual increase of vaso-motor tone could be demonstrated. Catheterization, massage of the drum-head and the removal, when found, of any causal agent was followed by relief chiefly in those patients with trouble of short duration; such patients, it is worthy of remark, all having blood pressure within the normal range. As a whole the number of cases is too small to be taken as indicative of any absolute statement of value; each case proving more or less a law unto itself and the necessity of forming several classes to meet certain qualifications has given subdivisions with so few examples of each that many more must be added to substantiate the factors indicated by the few.

THE OTOSCLEROTIC CASES.

As a symptom of otosclerosis tinnitus aurium if not the most serious factor is by far the most exasperating; in these cases in which objective signs are so often absent and in

* A Consideration of the Vascular Theory of Tinnitus Aurium. H. O. Reik.

which deafness progresses in spite of all measures of treatment, the clearing up of the etiology of even one of its manifestations may be of some service in making plain the whole. As a class these cases seem to offer a good field for further investigation of the cardio-vascular theory; as a part of the present report, though few in number they are not without interest. The diagnosis was made in all upon the history of slowly increasing deafness, absence of any well marked tympanic changes, increased bone conduction with reduction, by air, in the perception of the low notes of the scale and shortened or negative Rinne with the 512 v. s. fork. Regarding the relationship between tinnitus and blood pressure the results were as follows:

No.	Age.	Sex.	B. P.
27	33	M.	95 to 110 mm. of Hg.
37	32	M.	120 mm. of Hg.
38	23	F.	123 ,, ,, ,,
40	30	M.	110 ,, ,, ,,
43	25	F.	105 ,, ,, ,,

Patient No. 27 was under constant observation for several months and all methods were tried to relieve his tinnitus, which was continuous, synchronous with his heart beat and most distressing in pitch. Many blood pressure readings were made, the highest being 113, the average 100 mm. of Hg. His hearing remained the same, the reduction in his perception not being sufficient to incapacitate him for ordinary conversation so long as words were pronounced clearly.

Patient No. 43, with a tension approaching the low limit, had continuous tinnitus, but the note was soft in quality.

Patient 40, with Schwartze's symptom well marked, indicating as it does a dilatation of the vessels around the promitory, and with a arterial tension of 110 mm. of Hg., had a humming tinnitus which was not particularly annoying, his hearing, however, was fast failing.

Patients Nos. 37 and 38, with their blood pressures well within the normal range, were not bothered by tinnitus except at rare intervals.

These cases though proving nothing in themselves, show the existence of an inverse ratio between the low blood pressure and the degree of tinnitus which is to say the least suggestive.

There remains little more to be said, a general summing up seeming hardly of any greater value than the qualified conclusions reached after the discussion of the separate groups of cases. The findings in each individual case may be seen by referring to Chart II. The blood pressure records of the middle ear cases and the manner of classifying the same are shown in Chart I.

It must be remembered that no index of systolic blood pressure can be considered an absolute value, and whereas most of the measurements given come within the normal range, the markedly high were accounted for by some general disease.

Generally speaking the series of cases has indicated:

1st. That whereas most of the patients had normal arterial tension the measurements approaching hypotension were influenced by factors which could they have been eradicated might have shown these patients to have had an even lower index than that indicated.

2nd. That irritation of the canal and tympanum causes a reflex vaso-motor depression.

3rd. That in patients with tinnitus an increase of vascular pressure is accompanied by relief of the ear symptom.

4th. That the tinnitus aurium is most annoying and the hardest to relieve in those patients with the lowest blood pressure.

Finally, I wish to thank Dr. H. O. Reik for his guidance and encouragement, Dr. Theodore C. Janeway, of New York, for his opinion concerning the comparative values of blood pressure measurements and Dr. Roger S. Morris, of the Johns Hopkins Hospital, for many valuable hints concerning the estimation of general systolic blood pressure.

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A NOTE ON BRAIN ABSCESS FORMATIONS WITH REPORT OF CASES.*

By S. MacCUEN SMITH, M. D., Philadelphia, Pa.

It was my privilege quite recently to exhibit two of the following cases, together with the brain of the third, showing the abscess formations, before the Section on Otology and Laryngology of the College of Physicians of Philadelphia, at which time the two first-mentioned cases had completely recovered. This convalescence was especially noteworthy in the first patient on account of the marked improvement not only in her general health, but more especially in her mentality.

Case I. M. K., girl, aged seven years. When 18 months old she had measles, complicated by pneumonia. During convalescence she suffered from an otitis media of both ears. The right one recovered in due course, but the left ear continued to discharge uninterruptedly for about five years. The patient was first seen by me in March, 1909. She was poorly nourished, very irritable, had mental hebetude, and suffered from an extremely offensive otorrhea that was constant. From being bright and attentive at school, she had become dull and backward in her studies and could not even do an errand correctly. For instance, if sent to the store for two articles, she would return without anything, having forgotten what to ask for, or else would obtain something wholly different from that for which she was sent.

An examination showed the left ear to be filled with a brownish-yellow, foul-smelling discharge. Its removal revealed an entire absence of the membrana tympani, malleus and incus. There was marked sagging of the superior and posterior wall of the external auditory canal. The discharge

*Read by title.

escaped from the attic almost as fast as it could be cleared away with a cotton-carrier. There was some tenderness on deep pressure over the mastoid process, which, together with the bulging of the superior and posterior wall, and the streptococcic infection shown by cultures, induced us to advise a mastoid operation for the cure of the chronic otorrhea. No intracranial lesion was suspected, although the fact that the patient suffered from considerable loss of memory and increasing irritability was deemed a suggestive symptom.

First Operation. A radical operation was performed on March 16, 1909. During its progress a large, carious opening was found in the tegmen antri, through which a quantity of pus continually oozed. The introduction of a probe showed the location of the abscess formation to be in the temporal lobe, the sinus leading therefrom being sufficiently large to admit of a good-sized drainage tube.

The patient progressed exceedingly well following this operation. Her general condition seemed to improve for a period of about two weeks, when she was suddenly attacked by headache, frequent vomiting and violent convulsions, together with right lateral nystagmus of both eyes, the movements being at the rate of about one hundred a minute, while the right side of the face was drawn up rapidly succeeding spasmodic twitchings. Her temperature had been normal since her admission to the hospital until this time, when it rose to 101°, but again returned to normal three days later.

Second Operation. A second operation was immediately performed. The old tract was carefully explored but no additional pus was found there. The Jackson brain forceps were then introduced at a point posterior to the parent abscess formation and a large quantity of pus was evacuated from this locality. The patient at once improved but did not seem to fully regain the apparent state of health that followed the first operation. On the contrary, she continued to have occasional attacks of nausea and headache. Her temperature ranged between normal and 101° for five

CHART No. I.

Pressures from 100, or below, to 127					Pressures above 127				
No.	Age	Sex	Pressure	Considered	No.	Age	Sex	Pressure	Considered
3	25	F	125	Normal	1	28	F	135	Normal
4	21 28	F	120	Normal	6	39	F	135	Normal
5	25	F	120	Normal	20	45	F	140	Normal
15	54	F	125	Low	31	19	F	130	Normal
16	32	F	120	Normal	2	41	F	142	Normal
32	40	F	115	Low	13	37	M	145	Normal
34	44	M	115	Low	24	34	F	165	High
42	57	M	110	Low	39	43	F	180	High
46	42	F	120	Low	7	28	M	130	Normal
51	32	M	115	Normal (?)	10	42	F	135	Normal
8	18	F	125	Normal	18	36	F	135	Normal
9	35	F	115	Normal	29	36	M	135	Normal
33	25	F	120	Normal	47	26	M	130	Normal
44	71	M	127	Low	25	53	F	133	Low
52	49	M	115	Low	26	50	F	140	Normal
					48	50	F	130	Low (?)
					17	39	F	130	Low (?)
					11	30	M	130	Normal

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days following this second operation, and then remained stationary at normal. About four weeks after the second operation the frontal headache became continuous. The child was evidently very ill, and additional surgical interference was decided upon.

Third Operation. A third operation was performed April 26th, with the purpose in view of locating and evacuating any additional pus formations that might possibly be present This time the brain cortex was explored anterior to the parent abscess, and fortunately another pus sac was evacuated. No additional discharge was found escaping from either the first or second pus cavities.

April 28th, two days after the third operation, the patient was inoculated with a 50,000,000 (dead) staphylococcus aureus auto-vaccine obtained in pure culture from the pus evacuated from the brain. The inoculation was followed by a one degree rise of temperature within the next twenty-four hours, but there were no other apparent signs of systemic reaction. The immediate effects of the procedure were (1) marked slowing of the pulse rate; (2) increased output of urine; (3) normal temperature range; (4) cessation of vomiting and progressive disappearance of the aural discharge. The patient's opsonic index to the germ isolated remained high (1.1).

The child had at this time become extremely emaciated and weighed not more than twenty-five pounds. She gained an additional eighteen pounds before her discharge from the hospital, about three weeks after the last operation. Today, nearly one year later, she weighs about sixty pounds. Her memory is again retentive and accurate and she is doing well in school. There has been no recurrence of the pus symptoms. On account of the large opening in the bone, a hernia cerebri resulted, but it was confined within the bounds of the mastoid cavity. Notwithstanding the hernia and the radical mastoid operation performed on this ear, the child can still hear a low voice speaking at some distance.

It will be noted in this case that the patient did not at any time, either before or during her illness, have a sub-

normal temperature or subnormal pulse; furthermore, although she suffered apparently from three distinct abscess formations, the characteristic symptoms incident thereto were practically nil, showing that for a time, at least, they must have been small and encapsulated, and encroached only upon the so-called "silent area" of the temporal lobe; indeed, those pus sacs situated anterior and posterior to what we have seen fit to call the parent abscess formation may have been encapsulated at the time of the first operation. The subsequent symptoms, however, requiring the second and third operations, would seem to indicate some disturbance of the pus formation situated in the satellite abscess cavities. It would appear, also, that these three abscess formations were entirely isolated from each other, or else they would have drained through the outlet provided for the primary or parent abscess. If this had happened, the subsequent symptoms, requiring the second and third operations, could not have occurred.

It is altogether likely that this patient might have recovered without the use of the auto-vaccine, from the fact that we were fortunate enough to locate the pus and evacuate it. However, I wish to take this opportunity of expressing my belief that the employment of the vaccine was of distinct value and may have contributed largely not only to the patient's rapid recovery following the third operation, but probably inhibited further pus formation.

Case II. W. S., male, aged 37 years. When ten or twelve years of age he had an attack of scarlet fever, complicated by a suppurative otitis media of the left ear, which has been recurrent ever since, a period of about twenty-five years. The longest period between attacks was from two to three years. During this time he maintained a state of good health. On two different occasions, ten years or more ago, his physician, Dr. W. S. Jones, of Camden, removed a polypoid mass from the ear.

Examination showed the left ear to be well filled with a brownish-yellow, foul-smelling discharge, notwithstanding it had been cleansed thoroughly about an hour before. This

discharge was escaping through a large perforation in the postero-superior quadrant of the drum-head, involving almost one-half of this part of the membrane. The tympanic cavity was well filled with a large mass of granulation tissue. Considerable discharge was also escaping through a large, carious opening about the middle of the posterior osseous canal, through which a probe was passed directly into the antrum and revealed much carious bone. He suffered from attacks of vertigo about once in each week, of sufficient severity to cause him to fall unless he promptly received some support.

The patient believed that he was entirely deaf in this ear, although he could hear the tuning fork in close proximity to the concha. Bone conduction was greatly reduced from normal. No tenderness was elicited on pressure over the mastoid. On account of his apparent good health and in consequence of absence of suspicion of intracranial lesions, the usual preliminary examinations of blood and eye-ground were not made.

Operation. The radical mastoid operation was performed at the Jefferson Hospital, March 4, 1909. The cortex was found to be very thick and hard, with the exception of the mastoid tip, which was soft and necrotic. The antrum was reached with difficulty on account of the dense, eburnated bone. As soon as the latter was opened, a quantity of pus gushed out as though it had been under some pressure. When this was cleaned away the pus was seen to escape through a carious opening in the tegmen antri and through the dura. A probe was introduced through this opening into the interior of the skull, the sinus apparently leading into the temporal lobe. A pair of Jackson brain forceps were then introduced and separated, which permitted the escape of additional pus. Proper drainage was provided, and the patient made a slow but uninterrupted recovery.

The chief point of interest in this case is that the patient apparently suffered no inconvenience at any time from this pus formation located within the interior of the skull, unless we may attribute his spells of vertigo to this cause. It

would, of course, have been most interesting to have had a record of his visual fields and a differential blood count.

The ear at this time, after the lapse of a year, looks perfectly well in every respect, complete dermatization having taken place. He hears the tuning fork at about six inches, which is a gain of more than five.

Case III. C. M., white, male, aged 23 years. Suppuration of the right ear occurred when one year old, the discharge continuing for two years. From that time he was free from any aural trouble for twenty years, or until the present condition developed. This attack began three weeks prior to the first consultation, with severe pain in the right ear, followed in twenty-four hours by a discharge, which continued for three days and then suddenly stopped, the patient being immediately seized with severe pain in the back part of the head. This pain persisted for two weeks, when he consulted his attending physician, who immediately referred him to the Jefferson College Hospital.

The patient claimed he was unable to hear, but tests showed that he retained some sound perception through aerial conduction, as well as very good bone conduction. The canal of the right ear was filled with a creamy, foul-smelling pus, and was somewhat swollen. There was bulging of the superior part of the membrana tympani and the posterior wall of the canal, together with great tenderness on pressure over the process. Temperature was sub-normal, 97.1°.

The spontaneous opening in the drum-head was enlarged and the tympanic cavity aspirated, the patient being admitted to the ward for further examination and observation.

Prof. Howard F. Hansell examined the eyes and found them normal in every respect, except that the retinal veins were overfilled and tortuous, showing some interference with the venous circulation back of the eyeball. This congestion was more marked on the right side than on the left. The ocular examination, however, gave no indication of the site of the supposed cerebral lesion. Furthermore, the

neurological report of Prof. F. X. Dercum revealed nothing of importance in the way of localizing symptoms.

Prof. John H. Gibbon examined the case from a general surgical standpoint and reported that, in his opinion, the patient had an edema of the meninges and was suffering from intracranial pressure, but could find no evidence of a localized abscess. The patient died soon after admittance to the hospital.

The post-mortem examination revealed an abscess in the right temporal lobe, extending in the long axis. This was filled with very thick, yellowish-gray pus. Somewhat anterior to the center of the lobe was a second small abscess directly internal to and on a level with the larger one.

Here again it will be seen by actual post-mortem findings that the satellite abscess formation had positively no connection with the parent abscess, there being a considerable space of healthy brain separating the two. In the experience of Heinrich Neumann these abscess formations are usually connected by a sinus one with the other. Nevertheless, in the post-mortem examinations that have come under my notice, I have never found them connected with each other in this manner, but have noted that the foci were always isolated. The two cases reported in this paper would seem to still further confirm this view.

From the fact that brain abscess formations almost invariably complicate the chronic form of aural suppuration, we, as aurists, should be insistent in our endeavor to impress upon the profession the necessity for due recognition of this fact. Intracranial suppuration becomes impressively important when we realize that abscess of the brain may develop from the acute form of middle-ear disease without rupture of the tympanic membrane. Then, again, if we wait for focalizing symptoms, or definite diagnostic data of the abscess' existence and location before operating, it is usually too late to institute successful surgical intervention.

Another point to which I wish to direct attention is that a vast majority of all abscess formations involving the interior of the skull are not even suspected before opera-

tion, but are revealed at the time of a mastoid exenteration for a chronic otorrhea—and this applies equally to both intra and extra-dural suppurations. If, therefore, during a mastoid operation, we find the osseous structure, more especially the tegmen antri or tegmen tympani, discolored or otherwise diseased through carious erosion, or if a fistula is found leading into the brain substance, this unquestionably is our best and most natural route for exploration. A counter-opening in such cases is usually not only unnecessary but unwise, and in my judgment may cause additional grave complications. All of the cases of this character that have come under my observation have made good recoveries, chiefly, I believe, because of the good natural drainage that this route provides.

When the abscess is deeply situated, the infection being transmitted through the blood vessels or lymphatics, and no evidences of communication are found during the mastoid operation, a prompt external operation is not only justified, but demanded.

VACCINE THERAPY IN OTOLOGY.

By B. ALEX. RANDALL, M. A., M. D., Philadelphia.

The applications of opsonic treatment by vaccine, autogenous or standard, to the diseases of the ear have been too limited in number and apparently by too few investigators to have reached definite results. Hardly a hundred individual cases are on record and these, while encouraging, especially to the enthusiastic, have too many loopholes for overestimate to carry conviction to cooler minds. Anyone who has watched or shared in the rise and decline of numerous lines of treatment can recognize the same story—the citation of small series of successes, rather too invariable to be unselected; but probably feels that he could by several other methods fairly match them. The permanence or uniformity of success which should distinguish a fundamental improvement in our therapy cannot yet be considered proven; while some of the reporters lay themselves open to suspicion of being none too well-posted or else too pessimistic as to the older measures.

My personal experience has been limited; and this as much from distrust as from inertia. The field for such therapy is in the localized infections, which are generally open to the long-known and well-proven topical measures, which each of us can carry out unaided by the bacteriologist and "immunizator." When a generalized bacteremia is present and demands addition to our resources, the inoculations may be dangerous. Auto-inoculation is going on to the exhaustion of the opsonic or antitoxic powers of the organism, and we may add a fatal load to the already overtaxed individual. In more chronic cases, even when widespread, we may learn to profit by this line of medication; but serious results will be met, as from the induction of Bier's passive hyperemia, until by more generally reliable

determinations of the opsonic index, we make a surer discrimination of the unsuitable cases.

In tuberculous patients marked change for good has promptly showed in unprogressive or retrograding wounds; and I can warmly recommend the method even for cases under suspicion. The effort should be rather to secure the benefit as clinically appearing without reaction, than to use it like a von Pirquet test to get confirmation of the diagnosis. Most of the testimony even of some of the expertest "immunizators" has been against the usual clinical value of opsonic determinations; the difficulties limiting such tests to the experts with abundant time; while the temperature and other indications commonly suffice to guide the administrations. Yet in the cases of mixed infection the differences shown in the opsonic index for each organism may be of great value in selecting the vaccine or the combination and proportion from which most value may be expected, as also in warning against the inoculation at the very time when spontaneous auto-inoculation may have caused a marked negative phase. In these mixed cases, however, combined vaccines are claimed to be generally indicated either simultaneously or in alternation; and in the relapses new cultures should at once be made to learn if a new infection may not have been engrafted upon the cases, just as the immunity for the earlier caustive germ had been established.

It is not a violent supposition that most of the acute aural inflammations to which vaccine therapy has thus far been applied were practically self-limiting and would have healed under gentle topical medication and good tonics. So, too, every aurist of experience must have seen cases of very chronic, fetid and disquieting suppuration which had improved little or not at all under the care (real or alleged) of competent colleagues, yet became odorless or even dry under one or two treatments scrupulously applied, be the chosen medicament what it may. These are rarely free from backsliding at no very distant day; yet with renewed and prolonged success as a rule later. Since operative intervention has been so fashionable, such results have probably

been less wide-spread and frequent; yet the fault may not have been with the simple methods but with their less usual or half-hearted employment.

Without going into a detailed, critical analysis of the cases published by different observers or the larger number reported in private communications, it may be stated that a large proportion of the acute cases showed cure only in fair accord with the usual course of the disease, so far as an outsider can estimate it. In those presenting a more obstinate or chronic tendency, whether from more virulent infection or from depraved health of the individual, the response to vaccine therapy, which was usually combined with local measures, has been tardy, incomplete or doubtful. In the chronic cases the results have been even more unsatisfactory; but this may be discounted in part because it was often used as a last effort before resorting to needed operation.

Closer scrutiny of the instances of its employment will probably give to each student of the matter a greater confidence in the possibilities of the method; and it is to be hoped that this joint consideration will serve to crystalize into more available shape its practical applications, and that growing experience will show more frequent realization of the hopes and claims of the enthusiasts.

VACCINE THERAPY IN OTOLOGY.

By HENRY O. REIK, M. D., Baltimore, Md.

The scientific application of vaccine therapy, after the method suggested by Sir Almroth Wright, is of such recent origin that no one can be said to have had a large experience in its employment as yet, and, in so far as otology is concerned, the entire number of cases reported in medical literature is still too small to permit of drawing any very accurate conclusions regarding its value. If I understand the purpose of this meeting today, however, it is desirable that I should give you, rather than personal experience, a brief summary of what has been accomplished by the use of vaccines in otology, and, by deductions therefrom, attempt to forecast the future applicability of this new measure to the treatment of diseases of the ear. I shall therefore give little or no consideration to the underlying principles of vaccine therapy nor to the methods and technique of preparing and administering the remedy, assuming that you are all familiar with these points and confining my remarks as nearly as possible to the clinical features.

One of the earliest cases treated in this country, they believe it to have been the first, was reported by Magruder and Webb, of Colorado Springs. It was an interesting case of acute suppurative otitis media in a patient 42 years of age, first seen January 24, 1907. There was spontaneous rupture of the tympanic membrane and a pure culture of pneumococcus was obtained from the pus. The affection resisted all forms of treatment for 61 days, at which time there were symptoms indicative of commencing intracranial complications. The opsonic index for the pneumococcus was .7 and an autogenous vaccine was prepared. Two injections were given, the first of 14,000,000 killed micro-organisms, and the second, four days later, of 20,000,000.

CHART No. II.

No.	Sex.	Age.	Ear R. or L.	Duration and Character of Tinnitus.	General Health.	Appearance of the Tympanic Membrane.	Hearing Tests.	Diagnosis.	Etiological Factors.	Blood Pressure.		Remarks.
1	F	28	Both	Several years.	Good.	Both dull and slightly retracted.	Hearing not appreciably affected. Some reduction in the duration of the 128 v. s. fork.	Ch. Ex. Otitis Media.	Spur removed from septum. Inf. Turbinectomy both sides.	135 m. m. Hg. 125 " " " 135 " " " 130 " " " 135 " " "		Pt. typically neurasthenic. Hyperdermic of Suprarenalin Synthetica, grs. 1-200. Fleeting rise from 5 to 10 m. m. No improvement after prolonged treatment.
2	F	41	Both	6 months.	Good.	Both dull and slightly retracted.	Slight loss of perception for the 128 v. s. fork.	Ch. Ex. Otitis Media.	Middle Turbinectomy.	160 m. m. Hg. 150 " " "	5 c. m. band. 10 c. m. band.	Suprarenalin Extract increased the tinnitus and had to be discontinued. Some relief from catheterization and bromides.
3	F	25	Both	18 months.	Good.	Slight retraction. Scar in R. drum-head.	Reduction in lower scale.	Ch. Ex. Otitis Media.	Septal spur. Bad tonsils.	125 m. m. Hg. 130 " " "		Relieved by use of catheter.
4	F	28	L	3 months.	Good.	Retraction.	Reduction in lower scale.	Ch. Ex. Otitis Media.	Obstruction requiring use of catheter from R.	120 " " " 125 " " "		Relief from catheterization.
5	F	25	Both	Several years.	Good.	Slight retraction. Loss of light cone.	Loss in the lower scale.	Ch. Ex. Otitis Media.	Hypertrophied Inf. Turbinates at times. Impossible to use catheter.	120 m. m. Hg.		Tinnitus periodic. Relieved greatly by the use of the catheter and massage of the drum-heads.
6	F	39	Both but more marked in L	2 years.	"Nervous." "Heart Trouble."	R. Retracted and dull. L. Markedly retracted.	Well defined middle ear deafness. Cannot hear ordinary conversation.	Ch. Ex. Otitis Media.	None.	135 m. m. Hg. 140 " " "		Prolonged treatment with the catheter gave slight relief at times. Use of suprarenalin did not give definite results.
7	M	28	R	2 years.	Good.	R. Dull and retracted. No light cone. L. Slight retraction, partial light cone.	Reduction in lower scale. Weber to R.	Ch. Ex. Otitis Media.	Tonsils were removed.	130 m. m. Hg.		Prolonged treatment has improved hearing slightly and kept it from progressing. No definite results from the use of suprarenalin extract.
8	F	18	Both	5 months.	Good.	R. Atrophic, Calcareous deposits. Retracted. L. Atrophic. Slight calcareous deposits.	Watch. R. 6-12 inches. L. 6-12 inches.	Ch. Ex. Otitis Media, following "Scarlet Fever."		120 m. m. Hg. 123 " " " 125 " " "		Right ear slightly improved but no benefit in the left. Hearing not improved.
9	F	35	Both but more marked in left	5 months.	Good. "Nervous."	R. Retracted. Scar and calcareous deposits. L. Marked retraction.	Watch. R. 1-12 inches. L. c-12 inches. Definite loss in the lower notes and for "conversation."	Ch. Ex. Otitis Media.		115 m. m. Hg. 123 " " " 130 " " " 130 " " " 125 " " "		Suprarenal Rx. Feels better. Nearly free of noises. Suprarenalin by hypo. grs. 1-200, "fleeting" rise. Feels quite free from the tinnitus.
10	F	42	Both	2 months. Worse at nights.	Good.	Both show a very slight retraction.	Watch. R. 6-12 inches. L. 2-12 inches.	Ch. Ex. Otitis Media.	Nasal obstruction on right side.	135 m. m. Hg.		Improved by the use of the catheter and bromides.
11	M	30	Both	2 years or more.	?	R. Markedly retracted. L. Markedly retracted. Calcareous deposits.	Watch. R. 6-12 inches. L. 6-12 inches.	Ch. Ex. Otitis Media.		133 m. m. Hg.		Under observation in the Johns Hopkins Ward. Suffers with severe headaches, for which no reason could be found.

CHART No. II.—Continued.

No.	Sex.	Age.	Ear R. or L.	Duration and Character of Tinnitus.	General Health.	Appearance of the Tympanic Membrane.	Hearing Tests.	Diagnosis.	Etiological Factors.	Blood Pressure.		Remarks.
12	M	20	Both	8 months.	?	Normal.	Normal.	No disease.		115 m. m. Hg.		Has been listless and weak for eight months. Came to Hopkins Hospital for thorough physical examination. No organic lesion discoverable.
13	M	37	Both	3 months.	Good.	Both drum-head markedly retracted.	Watch. R. c-12 inches. L. c-12 inches.	Ch. Ex. Otitis Media.	Obstructed Eustachian tubes.	145 m. m. Hg.		Tinnitus improved by the use of the catheter and massage of the drumheads.
14	F	53	R	? Worse at night.	Good.	Retracted and dull.	Watch. R. 4-12 inches. L. 12-12 inches.	Ch. Ex. Otitis Media. Tubal obstruction.	R. Tube swollen and difficult to catheterize.	130 m. m. Hg.		
15	F	54	Both	?	Good. "Nervous."	Both. Retracted and dull.	Watch. R. c-12 inches. L. c-12 inches.	Ch. Ex. Otitis Media.	Atrophic Rhinitis.	130 m. m. Hg. 140 " " " 125 " " " 130 " " "		Rx. Suprarenalin Extract. Feels better. No better. Slightly better. Improvement not permanent.
16	F	32	Both	?	Good. "Nervous."	Both. Retracted and thickened chronic congestion along handle of malleous.	Watch. R. 6-12 inches. L. 4-12 inches.	Ch. Ex. Otitis Media.	Atrophic Rhinitis.	120 m. m. Hg. 80 " " " 90 " " " 120 " " "		Canals filled with wax. Syringing, patient fainted. Blood pressure drop.
17	F	39	Both	R. 5 years. L. 2 years. Worse in damp weather.	Good.	R. Thickened & retracted. L. Thickened & retracted.	Watch. R. 0-12 inches. L. 0-12 inches. Rinne negative Deafness for conversation.	Ch. Ex. Otitis Media.		130 m. m. Hg. 130 " " " 130 " " " 135 " " " 140 " " "	Over sleeve. Bare arm.	Catheter and R. Suprarenalin Extract. Feels better. Better. Much better. Can sleep well now. No tinnitus. Worked hard all the morning.
18	F	40	L	1 year.	Good.	R. Retracted. Calcareous deposits. L. Retracted and thickened and congestion along handle.	Watch. R. 0-12 inches. L. 8-12 inches. Weber to L.	Ch. Ex. Otitis Media.		135 m. m. Hg. 150 " " " 135 " " "		Rx—Suprarenalin. Much better. Feels worse. Some permanent benefit from the use of catheter.
19	M	33	L	?	Good. "Nervous."	R. Normal. L. Slight thickening.	Normal.	No ear trouble.	Deviation of nasal septum.	145 m. m. Hg.		Very large, muscular man. Has been a prize fighter. Report from Neurological Department—"Neurasthenia."
20	F	45	Both	2 months.	Good.	Both tympanic membranes retracted and thickened.		Ch. Ex. Otitis Media.		140 m. m. Hg. 145 " " "		Improvement rapid after the use of the catheter.
21	M	35	Both	2 weeks.	Good.	Normal.	Normal.	Impacted Cerumen.		135 m. m. Hg. 125 " " "	Before syringing. After syringing.	No further trouble.
22	F	20	Both	?	Good.	Drums slightly dull and thickened.	Reduction in perception of lower notes.	Cerumen. Sub-acute Ex. Ot. Media.		120 m. m. Hg. 110 " " "	Before syringing. After syringing.	Pt. treated afterwards for "stuffy" feeling and slight pain in ears. Tinnitus not present. B. P. taken to determine effect of syringing.
23	F	18	Both	Pain, but no tinnitus.	Pulmonary Tuberculosis	Normal.	Normal.	Impacted Cerumen.		100 m. m. Hg. 100 " " " 115 " " "	Before syringing. After syringing. 10 minutes later.	Pt. did not return, but heard from her sister that there had been no recurrence of the trouble.

CHART No. II.—Continued.

No.	Sex.	Age.	Ear R. or L.	Duration and Character of Tinnitus.	General Health.	Appearance of the Tympanic Membrane.	Hearing Tests.	Diagnosis.	Etiological Factors.	Blood Pressure.		Remarks.
24	F	37	R	Shooting pain and feeling of foreign body.	Interstitial Nephritis.	R. Drumhead retracted. Ant. half. M. T. movable malleous adherent to inner wall. "Black" spot in post-quadrant.	Watch. R. c-12 inches. L. 12-12 inches.	Fibrous adhesions in middle ear.	Suppurative Otitis in childhood.	165 m. m. Hg.		Nephritis for 8 years. When "black" spot in drum is touched there is a metallic taste in the mouth.
25	F	53	Both	"At times."	Alcoholic.	Both drums retracted and thickened.	Loss in perception of the lower notes.	Ch. Ex. Otitis Media.	Ethmoiditis.	133 m. m. Hg.		No relief. Pt. slightly under the effects of alcohol when the B. P. was taken.
26	F	50	R	1 year.		Both drums thickened and retracted.	Watch. R. 6-12 inches. L. 6-12 inches.	Ch. Ex. Otitis Media.		140 m. m. Hg.		
27	M	32	Both more marked L	Worse in damp weather. Continuous and pulsating.	Good.	R. Thin. Can see incus. (Long process). L. Thin. Can see incus. (Long process).	R. 6-12 inches. L. 6-12 inches. Bone conduction increased. Rinne shortened.	Otosclerosis.	Inf. Turb. removed. No nasal trouble. Tubes well open.	95 105 100 110 100 103 100 100 113 110	Pressure taken at intervals over several months.	Suprarenalin Ext. had no effect. Suprarenalin by hyperdermic. No effect. Fibrolysin. No effect. No benefit from any line of treatment used.
28	F	37	Both	2 weeks.	Good.	R. Congested and thickened, particularly membrana flacida. L. Congested & thickened.	Loss in perception of low notes.	Acute Ex. Ot. Media.		130 m. m. Hg.		Ear later suppurated and was treated by irrigations. The tinnitus ceased with the stopping of the discharge.
30	F	34	Both or L	Several weeks. Worse at night.	Good.	Slight retraction. Good light cone in both drumheads.	Watch. R. 12-12 inches. L. 12-12 inches. Forks v.s. 512. R. 30-30. L. 15-30 air Weber referred to the Left.	Exudative Ot. Media. (Slight).	Tubes well open. No nasal or throat trouble.	120 m. m. Hg.		Improved with Bromides.
29	M	36	Both or R	? At all times.	Good.	R. Slight retraction. L. Normal.	Watch. R. 2-12 inches. L. 8-12 inches. Weber to R.	Ch. Ex. Otitis Media.		135 m. m. Hg.		Did not return for treatment.
31	F	19	R	1 year. Worse at night and like a bell.	Good. "Nervous."	Slight retraction. Both practically normal.	R. 12-12 in. (?). L. 12-12 in. (?). Weber referred to R.	Exudative Ot. Media. (Tubal).	Tubes partially closed. Large Inf. Turb. Degenerative tonsils.	130 m. m. Hg.		
32	F	40	Both or L	Recurrent. Worse in last 6 weeks.	Good.	R. Marked in-drawing of handle. Partial light cone. L. Handle markedly in-drawn.	Watch. R. 10-12 inches. L. 4-12 inches.	Ex. Otitis Media Ch.	Large submerged tonsils.	115 m. m. Hg.		Suprarenalin. Some improvement from R. and ear treatment, but not permanent.
33	F	25	Both	1 month.	Good.	R. Dull and retracted. L. More in-drawing with good light cone.	Watch. R. 7-12 inches. L. 3-12 inches. Weber to L.	Ch. Ex. Otitis Media.	Tubes well open. No nasal trouble. Family history of deafness.	120 m. m. Hg. 127 " " " 125 " " " 110 " " "	Over sleeve. " " " " " "	R. Suprarenalin. Feels better. Feels better. Had tinnitus again this morning.
34	M	44	Both	?	Pulmonary Tuberculosis.	Membranes retracted, dull and thickened.	R. e-12 inches. L. c-12 inches. Deafness for "conversation."	Ch. Ex. Otitis Media.	Large Inf. Turbinates.	115 m. m. Hg.		No improvement.

CHART No. II.—Continued.

No.	Sex.	Age.	Ear R. or L.	Duration and Character of Tinnitus.	General Health.	Appearance of the Tympanic Membrane.	Hearing Tests.	Diagnosis.	Etiological Factors.	Blood Pressure.		Remarks.
35	F	65	R	1 month.	Insane at one time.	Both. Slight retraction.	R. 0-12 inches. L. 12-12 inches. B. C. O— B. C.—Normal.	Labyrinthine Deafness.	Deafness and tinnitus, followed "concussion."	137 m. m. Hg.	Over sleeve.	No improvement.
36	F	59	R	Vertigo and tinnitus for some time.	Myrocarditis.	R. Handle slightly indrawn. Good light cone. Calcareous deposits. L. Retracted slightly.	R. Neg. to air and bone conduction. L. 6-12 inches.—Bone conduction normal. Weber to L.	Labyrinthine deafness. Ménière's symptoms.		175 m. m. Hg.	Over sleeve.	Tinnitus not severe. Some improvement with K. I. and tonic treatment.
37	M	30	Both	Tinnitus slight and only at times.	Good.	R. Normal. "Thin." L. Normal. "Thin."	R. 2-12 inches. Bone conduction normal. L. c-12 inches. B. C. normal. Lower notes lost.	Otosclerosis.	Tubes well open. No nasal or throat trouble.	120 m. m. Hg.		Hearing kept at one point.
38	F	23	R	2 months. Tinnitus only at times.	Good.	R. Slight retraction. L. Slight retraction.	Watch. R. 2-12 inches. L. 4-12 inches.	Otosclerosis.	Tubes well open. Chronic Naso-pharyngitis.	123 m. m. Hg.		Felt better after treatment of throat and ear.
39	F	43	R	1 week. Tinnitus and pain in R. ear.	Good.	R. Thickened & retracted. L. Thickened & retracted.	Did not test.	Ch. Ex. Otitis Media. with exacebation of sub-acute symptoms.	Naso-pharyngitis.	180 m. m. Hg. 190 " " "	First. Second a few minutes later.	Specimen of urine asked for, but patient did not return.
40	M	30	Both	R. 4 years. L. 10 years. Tinnitus constant, but not bad.	Good.	Both slightly in-drawn. Good light cone. "Pinkish" tinge in posterior part.	R. 0-12. B. C. 512 v. s.—15-15. L. c-12. B. C. 512 v. s.—15-15. Loss of low notes to 512 v. s.	Otosclerosis.	Synechia in nose from use of the cautery.	110 m. m. Hg.		Has been treated by many Otologists without relief. Tinnitus not annoying. Deafness increasing rapidly.
41	F	51	R	Some months. Extreme all the time.	Good.	R. Thickened and congested in membrana tacida. Canal irritated. L. Dull	R. c-12 inches. L. c-12 inches.	Sub-acute Ex. Otitis Media.	Chronic Naso-pharyngitis.	155 m. m. Hg.		℞. Carbolized glycerin 10%. Catheter and massage. Bromides and later Tinct. Nux Vomica. Complete relief after some weeks' treatment.
42	M	57	Both or L	7 months. Hissing and crackling.	"Neurasthenia."	R. Dull & slight retraction. L. Dull & slight retraction. No light cone.	R. 1-12 inches. B. C. normal. L. 1-12 inches. B. C. normal. Lower notes reduced in duration.	Ch. Ex. Otitis Media.	Large Inf. Turbinates.	110 m. m. Hg.	Hospital records show B. P. to be from 90 to 110 m. m. Hg. over a long period.	Mentality (?). Hears "click" in ears. Various disorders and complaints for which no cause can be found.
43	F	25	Both	Several years.	Good.	Both drums nearly normal. Diminution in light cone and slight prominence of malleous	R. 6-12 inches. L. 6-12 inches. Bone conduction increased. Weber to R. Low notes show loss.	Otosclerosis.		107 m. m. Hg.		Treatment not very satisfactory.
44	M	71	R	1 year. "Clicking."	Good.	Drums normal in appearance for age. Good light cone. Slight indrawing of handle.	R. c-12 inches. L. c-12 inches. High notes well heard. Good bone conduction.	Ch. Catarrhal Otitis Media.	Deviation of septum toward R.	127 m. m. Hg.		Improved slightly by use of catheter and Bromides.

CHART No. II.—Continued.

No.	Sex.	Age.	Ear R. or L.	Duration and Character of Tinnitus.	General Health.	Appearance of the Tympanic Membrane.	Hearing Tests.	Diagnosis.	Etiological Factors.	Blood Pressure.		Remarks.
45	M	35	R	2 months.	Good.	Both. Slight retraction and dullness.	Weber to R. Slight middle ear deafness.	Tubal Catarrh.	Tinnitus and deafness worse after child-birth.	110 m. m. Hg.		Pt. did not return.
46	F	42	Both or L	Some time.	Good. "Nervous."	R. Slight retraction. L. Retraction more marked. Thick and dull.	Middle ear deafness. Weber to Left.	Ch. Ex. Otitis Media.	Tubes easy to catheterize.	120 m. m. Hg.		Improved under treatment, but did not come regularly.
47	M	26	Both or L	Several years.	Good.	Both drums retracted and thickened.	R. 0-12 inches. L. c-12 inches. Deaf for "conversational tone."	Ch. Ex. Otitis Media.	Submerged tonsils.	130 m. m. Hg.		No marked improvement.
48	F	50	Both or R	Deafness for some years. Tinnitus recent.	Alcoholic.	Drums markedly retracted in upper part, due to in drawn malleous handle. Fair light cone.	Marked middle ear deafness, with secondary involvement of labyrinth in R.	Ch. Ex. Otitis Media.	No nasal or throat trouble.	130 m. m. Hg.		Marked mental element. Hears voices talking to her. Restriction of alcohol improved her condition. Tinnitus persisted, but there were no further "hallucinations."
49	F	36	Both	Some months. Occipital Headache.	?	Normal.	Normal.	No Disease.	Gynaecological Pt.	145 m. m. Hg.	Over sleeve.	Severe occipital headaches, with slight tinnitus. Unable to follow case regularly.
50	M	22	Both	?	Nephritis.	Both. Very slight change in drumheads.	R. Neg. by air and bone. L. Some perception for middle octave.	Labyrinthine Deafness.	Is under treatment for Nephritis.	140 m. m. Hg.		Albumin in urine. Says he had "fever" when a small boy, which left him deaf.
51	M	32	R	Vertigo at times. Tinnitus all the times.	Good.	R. Handle in-drawn. No light cone. L. Normal.	R. 4-12 inches. Slight middle ear deafness shown by loss in low notes. B. C. normal. L. Normal. Weber to R.	Ex. Otitis Media.	Large middle turbinates on R. side.	120 m. m. Hg. 125 " " "		Slight improvement after nasal operation and prolonged aural treatment.
52	F	49	Both	Very slight tinnitus now, but was very bad at first.	Good.	Both retracted and dull.	R. ½-12 inches. L. 0-12 inches.	Ch. Ex. Otitis Media.	Complete nasal operation for Pan-Sinusitis.	115 m. m. Hg.		No improvement in hearing. Tinnitus gives him very little trouble at present.

The discharge absolutely ceased and the ear was dry on the day following the second treatment; it remained well ten months afterward, when the report was published.

Tod and Western, in London, had applied the method to the treatment of tuberculous disease of the ear prior to that time, in one case as early as October, 1906. Their first case was one of chronic purulent otitis media of nine months duration. There was extensive glandular involvement and an open sinus in the neck. Mixed infection of tubercle with staphylococcus aureus. After two injections with tuberculin and staphylococcus vaccine alternately produced rapid healing. The second case was a youth of 20 years with tuberculous otitis media of six weeks standing. His treatment was administered somewhat irregularly but he eventually recovered. Case III was a four year old child with tuberculous disease of the ear and osteomyelitis of the tibia. After six months of the usual treatment a mastoid operation was performed. At that time there existed an offensive discharge, facial paralysis, enlarged glands, and carious bone could be felt by probe. Tympano-mastoid exenteration did not cure the aural affection, the discharge continuing, and, one year later, the opsonic index being .8 to the tubercle bacillus, inoculations were given. In three months time the aural discharge had stopped, the neck glands disappeared, the sinus from the tibia closed and only the facial paralysis remained.

Dr. Alice Hamilton, of Chicago, very carefully studied a series of acute aural infections associated with scarlet fever in one of the city hospitals. The patients submitted to vaccine treatment did well, but another group of the same number of patients in the wards did equally well under the routine treatment of cleansing. An interesting thing in her report is the finding of the pseudo-diphtheria bacillus in 72 per cent. of the 43 cases of acute scarlatinal otitis, and in 20 per cent. of these in pure culture. Here opsonic work and vaccine therapy seem to have been directed principally to this organism, one, by the way, that has generally been looked upon as of secondary importance and considered as

an accidental accompaniment of the pyogenic organisms that were actually doing the mischief.

At the annual meeting of the Central Section of the American Laryngological, Rhinological and Otological Society, on February 22, 1908, Joseph C. Beck described the methods of preparing vaccines and related a series of cases in which he had employed the method. This series included four cases of chronic purulent otitis media; two of double infection, pneumococcus and staphylococcus, one due to Friedlander bacillus and one of diphtheria. All were improved by the treatment but the published report does not contain detailed histories nor state the exact results.

Dr. W. W. Trimble, of Rosedale. Kansas, reported five rather interesting cases in May, 1908. The first case was one of chronic purulent otitis media following scarlet fever, the discharge having continued for two years; staphylococcus aureus was the germ present. Three injections of vaccine were given and in twelve days the patient was cured. The second case was one of nine years duration, staphylococcus infection, in which four injections sufficed to terminate the treatment. Case three was one of pyocyaneous infection, upon which treatment had no effect. A better result was obtained in the next pyocyaneous case, however, the staphylococcus albus being here associated with this germ. It was a chronic case, of two years duration, originating in an attack of scarlet fever. Three inoculations of a mixed vaccine caused disappearance of the pyocyaneous, and then a pure vaccine of the staphylococcus albus speedily produced a cure. The fifth case was one of less chronicity but greater virulence. A streptococcic otitis media of two months standing, with acute infection of the mastoid. There was a leucocytosis of 16,000, which was reduced to 12,000 after one injection of an autogenous vaccine, but a mastoid operation became necessary.

Dr. Trimble concludes his paper with an expression of his belief that "All cases of chronic suppurative otitis media in which symptoms are scarcely such as to justify (he probably meant demand) operating should be given vaccine

treatment. Failure will follow in many cases but so also does other treatment fail; on the whole this treatment has back of it good scientific principles and without doubt, when judiciously used will afford the greatest percentage of recoveries in a given number of cases."

Dr. Ray Connor, of Detroit, has tried the method in a variety of cases and in some rather discouraging conditions, yet, it seems to the reviewer, his results were very satisfactory, although his experience has not made him an enthusiast. The first six cases were of a most unpromising type, weak and miserably nourished babies, that would have done badly under any form of treatment. Four of these were cured; one was a streptococcus infection, one the Friedlander bacillus and staphylococcus, one staphylococcus aureus and albus, and another a mixed infection of diphtheria bacillus with pneumococcus. Of the two failures, one was a scarlet fever case and the other a mixed infection of streptococcus and staphylococcus. The average period of treatment was three months. A case of chronic purulent otitis, the infection being due to pyocyaneous mixed with staphylococcus, that had resisted all forms of treatment for two years, responded very satisfactorily to vaccines.

In the first case in which the vaccines were employed as after treatment to a radical mastoid operation, there was no apparent influence upon the healing process. The next two cases, however, in which the method was used as post-operative treatment, gave better results. The first was an epidural abscess which at the end of six weeks was not doing well; injections of micrococcus catarrhalis were given and the wound showed immediate improvement and was completely closed within three weeks. "Another similar case of epidural abscess was treated with an autogenous streptococcus and staphylococcus aureus suspension, beginning ten days after operation, but healed slowly and was not completely closed after three months treatment. When, however, the injections were stopped the progress of repair retrograded rapidly and the amount of secretion increased." "In still another simple mastoid repair went on nicely for

about two months, when progress seemed to stop. The wound, though small, showed little tendency to close. A pure culture of staphylococcus albus was found and a few injections of vaccine closed the wound rapidly and permanently in two weeks.''

Duncan, in a recent paper, reports Webb as having cured two more cases of purulent otitis media, this time the infective agent being the pneumococcus, with only two injections each. At the same time he relates a very unusual and interesting experience in the treatment of an infant suffering with double suppurative otitis media, due to mixed infection of pneumococcus and staphylococcus, that was resisting ordinary treatment. As the infant was nursing, inoculations were administered to the mother, with the result that the discharge from the infant's ears lessened and one was healed after the second injection. The infant was then inoculated and a complete cure promptly resulted.

C. L. McDonald, of Cleveland, in his observations upon 100 cases treated by vaccine therapy, included three cases of staphylococcus otitis media, with two cures, one of these having suffered a persistent otorrhoea for 30 years.

At the annual session of the American Laryngological, Rhinological and Otological Society, held in this city during the past week, Dr. E. W. Nagle, of Boston, presented a most satisfactory monograph upon the results of vaccine therapy in chronic suppurative otitis media. Dr. Nagle's work was of the most scientific character and her conclusions most convincing. If I understood her, and I have to give this from memory, she had treated 40 cases; in six of these the suppuration had existed only a few months, while the other 34 had been subject to otorrhoea for periods varying from one to forty years. Cure resulted in 39, the single failure being in the group of more acute conditions. She used autogenous vaccines prepared with the greatest care and the technique of treatment, as well, was conducted with skill and judgment. The micro-organisms found in her series of cases were chiefly the staphylococcus, influenza bacillus and bacilli of the proteus type. Dr. Nagle believes

that autogenous vaccines are preferable to stock preparations and emphasized the importance of killing the bacteria by the shortest exposure to the lowest temperature that would prove effective. This point was strongly endorsed by Dr. F. E. Sondern, the pathologist, of New York, who was inclined to believe the high value of her vaccines largely due to this factor.

Dr. F. C. Cobb, who had been in charge of some of the cases submitted to Dr. Nagle for vaccine treatment, and which were included in her report, testified as to the character of these infections and the fact that they were cured as described; he expressed the opinion that, in vaccine therapy "we have a very valuable adjunct to our armentarium."

In the course of a general discussion on this paper some rather interesting points were brought out. In the first place, Dr. J. C. Beck stated that he has now treated 74 cases of aural disease by this method but that his results have not been so good; only ten cases remaining well. He had, however, given the injections at intervals of from seven to ten days; whereas Dr. Nagle had given them at average intervals of three days, relying rather upon her judgment of the clinical evidences of the opsonic curve than upon any fixed periods for injection. Much of Dr. Beck's work had, furthermore, apparently been done with stock vaccines. A second point of discussion turned on the applicability of the method to the treatment of acute inflammatory conditions. The view generally held by Wright and other pathologists is that little would be expected from the use of this measure in acute infections because there is no apparent scientific reason for considering it appropriate. It may be well, however, to keep in mind the peculiar conditions attending some aural infections; a serious complication occurring to one who is the subject of long standing otitis media may be an acute infection, while, on the other hand, an otorrhoea that has existed but a very short time may have all the attributes of a chronic infection because the patient's ability to resist or to fight an infective process

is below par. Dr. Ewing W. Day reported six cases of acute streptococcic infections treated by vaccine with remarkably satisfactory results. Two of these were cases of infective lepto-meningitis; in one vaccines were given, in the other both vaccine and antistreptococcic serum. In one of his cases the vaccine was employed as post-operative treatment after a mastoid operation, when the patient was not doing well. Dr. H. S. Hedges also reported a case of staphylococcus otitic meningitis treated during the acute stage with vaccine, with an excellent result.

Now, it would be useless, if not indeed manifestly unfair, to try to draw definite conclusions from a series of abstracted case reports, such as I have presented here, from a number of operators working with varying types of infection and under varying circumstances. But it does seem fair to call attention to the positive results obtained by Nagle, Connor, Trimble, Magruder and Webb, McDonald and Tod, and to say, that their results afford considerable encouragement for further investigation. It must be kept in mind that the first application of this method to otology has been to the very worst class of cases and that the measure has been submitted to the most severe test. Tuberculous otitis media, the type that Tod dealt with, is notoriously difficult to heal; most of Connor's cases were of a desperate character, the condition of the children being such that an otologist might congratulate himself if he secured good results from any treatment whatsoever; many of the others refer to their treating cases of chronic suppurative otitis media that had resisted all other treatment for many years.

If we step outside the realm of otology and consider for the moment some of the recent work of general surgeons and bacteriologists experimenting with vaccines we shall learn several things of importance to those who contemplate trying the method in the treatment of diseases of the ear. In the first place, aural infections are not infrequently mixed infections; this is particularly true of chronic purulent otitis media. As to the proper course to pursue in such cases,

Olmacher says: "In a general way it may be said that a mixed infection offers a less promising outlook than that by a single bacterial species, and still some brilliantly successful results have been obtained by inoculation in very complicated and long standing infections. Given two, or even three, bacterial species, well known as pathogenic agents, and their simultaneous appearance in the secretions of a certain lesion, it is entirely proper to inoculate with a mixed vaccine containing proper doses of the offending bacteria. Or, when the urgency is not too great, inoculation with the predominating and most likely pathogenic agent is to be first performed and, in case of unsatisfactory issue, a vaccine from the other bacterial species can be added to subsequent injections. In dealing with mixed infections of a pyogenic nature, it is necessary to follow events in the more chronic suppurations by bacteriologic analyses from time to time, and to modify the inoculations to correspond with the changes of bacterial flora in the pus, in case the therapeutic response is not satisfactory."

Secondly, having to deal often with long existing localized inflammations of bone structure, we must not forget that such sinuses are frequently more or less encapsulated by inflammatory fibrous tissue, that their blood supply is poor and that, in consequence, they are not so easily reached by remedies through the circulatory system. We have no right to expect vaccines to remove sequestra, cholesteatomatous masses nor areas of bone necrosis. These must be removed surgically and free drainage of the sinus or abscess cavity afforded; the experience of general surgeons with stubborn abscesses and sinuses seems to indicate that the value of surgical measures may be greatly enhanced by the after use of vaccines, in fact, that surgical treatment is saved from failure by the assistance of this additional therapy.

Thirdly, there seems to be something for us to consider in Wright's original suggestion regarding the employment of vaccines preparatory to the introduction of surgical measures. On this point, Harris says: "I think the study of opsonins and bacterial inoculations teaches us very clearly

this fact, I mean those of us who are surgeons, that under certain conditions, particularly in tubercle, it is a wise thing to ascertain what the opsonic content is before operating, and, if it is low, and, if time will permit, to artificially raise it by inoculation before operating if we wish to obtain rapid healing.'' There is food for thought here and reason to hope that by following this advice we may secure a higher percentage of permanent successes in our tympano-mastoid exenterations. May we not hope that in such cases of chronic suppurative otitis media as cannot be cured by the vaccines their use will at least bring the patient to such a condition as to insure the success of an operation?

Fourthly, it seems to be the general consensus of opinion that the taking of the opsonic index is not an essential part of vaccine therapy, that its application requires a high degree of skill or much practice, that with anything short of absolute accuracy the results cannot be relied upon, and, that in so far as the treatment of a certain class of diseases is concerned clinical evidences are sufficient to guide one in the use of the vaccines.

Fifthly, that if used with reasonable care as to dosage and, especially as to asepsis, vaccine therapy is a harmless measure and carries with it practically no serious risks.

In bringing this résumé to a close, I should like to offer for your consideration the following conclusions and suggestions bearing upon the relationship of vaccine therapy to otology:

1. The employment of vaccine therapy in otology, although having had but a limited trial so far, has been attended by such successful results as to warrant us in feeling greatly encouraged.

2. Furunculosis, with its recognized tendency to the formation of boils in crops, seems to have responded more promptly and satisfactorily to this form of treatment than to any other, and, in tuberculosis of the middle ear, a condition heretofore so baffling to the otologist, there would seem to be good reason to believe that at least we have a controlling, curative remedy.

3. Looking into the future, we have held out to us the enticing prospect of being able to cure without operation a larger percentage of our cases of persistent chronic, purulent otitis media, or, failing in that, of insuring success for the operative treatment of this affection.

4. The aural conditions to which this new form of treatment would appear to be reasonably applicable are: the treatment of chronic suppurative otitis media; the preparation of patients for operation, raising the opsonic index in order to facilitate rapid healing after obliteration of the gross lesions; and, the post-operative treatment of mastoiditis and its complications, where healing is delayed and the patient is unable to construct new tissue without aid.

5. While the outlook is hopeful in these several directions, we must not grow too enthusiastic as yet. We are, perhaps, justified in some degree of optimism but not in enthusiasm. Let us try the method further, with fairness and judiciously, not expecting the impossible of it, for it will not entirely displace any other form of treatment; no matter how valuable it ultimately becomes, it will only be an additional therapeutic measure at our command.

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Discussion.

Dr. Solis Cohen, Philadelphia. I have had a very prolonged experience with tuberculin; for thirty years I was consulting physician for consumptives, and gave the first injection of tuberculin in Philadelphia before my class at the Jefferson Medical College upon a patient with advanced tuberculosis of the larynx in the early eighties. Ten years ago I heard that that man was well and was a successful ranchman in Wyoming. I have not heard from him since. Fifteen or sixteen years ago a man came to me from the south with his physician, with extensive ulceration of the larynx. He was placed under treatment and after a number of months his disease was arrested. Two years afterward his life was insured for $100,000 in the Mutual Life Insurance Company of New York. In that case not only was the injection given hypodermically but the tuberculin was rubbed over the laryngeal surfaces, and he took rectal injections himself every night. There is no doubt that in this case there was a great success. I know of several other cases still living at different periods of five to fifteen years or more, and doing well. The tuberculin that I have used has always been the old tuberculin of Koch, except when we were unable to get it, and then other forms were used, but did not appear to present any special advantages. These injections were used very much as the last two speakers have described, beginning with minute doses and gradually increasing them, and studying the patient rather than the disease.

I would like also to state something else. When I began the study of medicine some fifty-five years ago, I studied under a preceptor, as was customary at that time; and it was the custom then to apply fly blisters as near as possible over the site of the lesion of phthisis. We knew nothing of the bacillus tuberculosis at that time. Many of the cases improved, and since the tuberculin and serum treatment have been applied I have come to the conclusion that possibly the absorption of some of the serum raised by these blisters was beneficial in the same manner as the serum when injected under the skin, and acting on that principle, it has been my custom with many of my patients to blister the chest below the clavicle, taking care that the blister shall not burst, so that the patient's own serum shall be absorbed on the same principle as the autogenous methods. I do not often attend these meetings, but would like to give my testimony as to the advantage of this treatment in properly selected cases and under proper precautions.

Dr. Bowditch, Boston. These papers have been of intense interest to me and I trust that the discussion will be carried farther. I can rank myself in the first class spoken of by Dr. Miller, who tried it as soon as it was given to the public and after a few cases gave it up. I abandoned it after a few cases, largely influenced by the statement that there was danger of causing disseminated tuberculosis, and for many years I had a great repugnance against it. Finally I felt that I was doing injustice to many patients who might possibly be having treatment withheld which would do them good. Feeling with Dr. Griffin that certain cases had been going on under dietetic and hygienic treatment and making no progress, we agreed to give them the benefit of this treatment, and did so; the results were so startling that we felt justified in going on. To make a long story short we now, in addition to beginning with cases which have not been doing well, we begin on the incipient cases also. My position now is that of being greatly impressed with the tuberculin.

Dr. Griffin, Sharon, Mass. Taking up the consideration of Dr. Bowditch's last remarks, I would like to say something further about what we have been doing at the Sharon Sanitarium. We began with one case which had been to us before and under hygienic treatment for seven months, and had been discharged as an arrested case. In seven months she was back again, and although we kept her under hygienic treatment for five months she made no improvement beyond a certain point. It was this case which Dr. Bowditch said would be worth trying with tuberculin. We gave her treatment for five months, at the end of which time the tubercle bacillus had disappeared, and now she has been at times her laborious work in the kitchen. I saw her within a month past and went over her chest, and found her in very good condition.

The second case was similar in that the patient had been with us for several months and was not only not doing well, but was losing ground; the cough was increased and there were beginning objective signs in the larynx. She had tuberculin treatment for seven months, and it is her boast now that she has been out nearly five years and has not seen a doctor at any time within that period. We have now to report on twenty-eight cases which have had tuberculin treatment at Sharon and have been discharged for more than a year. Of these twenty-eight patients, six have died; two were really too far advanced to warrant advising tuberculin but took it at their own request; four others were advanced cases and were given tuberculin because it did not seem possible that they could improve under ordinary hygienic treatment, and it seemed to be a forlorn chance for them. Four others are not doing particularly well. They were all advanced cases, but I am afraid they are going down hill. This makes eighteen cases out of the twenty-eight which are doing well, and this seems to be a comparatively good record, when all of the cases with the exception of three were more than incipient, and all but the three had tubercle bacilli in the sputum. These three were

given treatment for special reasons. It is known that the tuberculin will do no harm and frequently will do good.

Dr. Nagle, Boston. I do not want to be too enthusiastic about vaccine therapy, but I believe it is a valuable adjunct in treatment. I wish, however, to emphasize the importance of the proper preparation of the vaccines and the method of giving them.

Dr. Minor, Asheville. The point is whether or not tuberculin should be used on every patient or only on selected cases. The tendency of some is to use it on every case. I have been slowly brought around from scepticism to belief in tuberculin, granted that it is a selective use. I don't believe that a patient should be put on tuberculin as long as he is making progressive and steady improvement; but for those who have done well up to a certain point and then reach a stage of chronicity when they go neither forward nor back, I believe that tuberculin is a valuable aid. When used in this way I have used it with very wonderful effects. I think if the idea went out that it was applicable to all cases, much damage would be done by it. To show how dangerous it may be when used promiscuously, I would like to cite the case of a boy seen by a doctor after he had been in the hands of a quack. The boy had incipient tuberculosis, and the quack started at once on daily injections of tuberculin; the boy began to have reactions, and the quack continued the treatment until the boy became a hopeless case. Men with dangerous and uncontrolled enthusiasm might do something of the same kind with it.

I believe that it is valuable when used selectively, but all cases do not do well on it, and some cases, with the greatest care are unable to take it. I would advise great care in the selection of cases for this treatment.

Dr. W. B. Johnson, Patterson. The discussion is developing along the lines of tubercular disease and tuberculin. I would judge that tuberculin has been successful in a number of cases, but the important consideration to the otologist

is what can be done with the autogenous or stock serum for the destruction of bacterial elements which enter into the diseases we are called upon to treat. I have followed serum and vaccine therapy for a number of years. One case of sarcoma of the pharynx I treated with the Coley serum with success—it was one of the few cases that were successful. I have also used the stock vaccines and serums, and while it seemed as if in some cases life was prolonged by the use of the serum and vaccine, it was not with me successful. It may have been that the preparations were poor. It has been stated lately that success has been attained by the use of stock vaccines, but in the cases I have known which have been limited to autogenous vaccines prepared in Jersey City, by Dr. McDowell, they also seemed to be unsuccessful. I do not wish to be understood as in any way depreciating the value of experiments in this direction, but I am still waiting and hoping that the results reported will make it desirable for all of us to use these remedial agents for the destruction of bacteria which enter into the diseases we have to treat.

Dr. Casselberry, Chicago. I have been particularly impressed with the faborable statement which has been given by one of the essayists, Dr. King, who refers to the pronounced favorable effects of tuberculin on laryngeal conditions in pulmonary tuberculosis, since we have not had up to this time any judgment or verdict in this respect based on so large an experience as he has had in this complication of tuberculosis. When the larynx is involved it becomes almost the dominant factor in the case, and our results up to this time have been discouragingly bad. Therefore, if we have got, as we have hoped for the last few years with the trend of the verdicts with respect to tuberculosis—if we have a remedy in tuberculin which will have in any considerable percentage of cases such effect as he has stated, we have made an enormous advance. I have been impressed with the statement that in those cases in which he got very pronounced reactions from a larger dose than was meant that the effect was good. This I have noticed

many times in making the tuberculin test when it was given for the purpose of producing a general reaction; I have noticed that weeks, and even months, afterward there has been marked improvement in the larynx. I noticed this, but did not trust to my own observation, but gave the credit to other circumstances, but the two go together; he has attributed it to tuberculin, and I have no doubt that he is correct. I have also had a limited experience with respect to the effects of the present day minute doses in laryngeal cases, in which recourse was made to all other medical and surgical treatment. Elsewhere, I have reported forty per cent. of arrested cases in laryngeal tuberculosis, some of them being those in which tuberculin in small doses had been given as an element of the treatment. Many of the men who see large numbers of pulmonary cases fail to examine the larynx. If these men would take note of the condition of the larynx and the effects on conditions there produced by tuberculin treatment, much good would be done to humanity in the direction of laryngopulmonary tuberculosis.

Dr. Dunn, Ashville, N. C. There are only one or two points in these very excellent papers which I wish to comment upon. First, I want to say that it is just such good, conservative papers as these which help. The tuberculin cause has been damaged more by optimists than by anything else. The effect of tuberculin in laryngeal tuberculosis has interested me for many years. I always examine the larynx, and I have been particularly struck with the improvement taking place in distinct laryngeal involvement. It is not so very striking in the advanced cases where there are marked destructive changes. As to the improvement following the reaction, it has been my custom to watch for reactions, and I have in many instances seen the beginning of improvement in the laryngeal reaction; it has happened so frequently that I am convinced it had some association with the reaction. As to the different forms of tuberculin, I have used a number of them; I used the old tuberculin constantly and exclusively for nearly ten years; for the last few years I have

used the human bacillus culture, and recently I have used the bouillon filtrate of Denis, and I have used other methods in vogue at times. I have used the B. E. and B. F. mostly of late, but the immediate results have been practically the same; I cannot see that I have gotten any better results from bacillus emulsion or from B. F. than from the old tuberculin. Not enough time has yet elasped to form a positive opinion as to the use of tuberculin. I am very conservative as to its value; on the other hand, I do not think our attitude should be that of giving a person the choice of taking it or not as he pleases. When I give tuberculin to a patient I expect him to take it, just as I expect him to obey when I tell him to go to bed, and if I am convinced of its usefulness, that is my privilege. As to waiting until the improvement under hygiene and diet have been tried, that is not fair; if we are convinced that it has a value in making results more permanent, it is our duty to our patients to do that which gives them not only the best immediate results but the best permanent chance. The time element is a very important one. I have known of a number of patients who took it for only a few months and then discontinued it and were put into a state of instability. That has been my impression.

Another thing: The greatest disadvantage in tuberculin is that the doctor puts his faith in his hypodermic syringe and does not pay enough attention to the life of the patient. We should never lose sight of the fact that it is a more or less important adjunct. Sometimes the patient puts more faith in what he gets from the hypodermic syringe than from what he does for himself. We should make them believe that what they do is of more importance than what we can do, and that the hypodermic is an aid, and nothing more.

Dr. I. H. Hanez, Lakewood. A few months ago I read a short abstract of an article written by a Frenchman—only a few lines, but in line of what has been said about the auto-inoculation—to the effect that in treating pleuritic effusions he drew 2 c. c. and then reinoculated his patient.

He reported four or six cases of value treated in that way. In view of this, I asked Dr. Trudeau if anything had been done at Saranac Lake with autoinoculation with the serum. It occurred to me that if that were drawn off as horses' serum is drawn it could be successfully used on the patient —the serum being drawn from his or her body. I am out of that work now, doing more general work, but I saw today two men who have opportunity to do that sort of bacteriological work. It is in line with what Dr. Cohen said—leaving the serum under the blister for reabsorption and autoinoculation that way.

Dr. Reik. I think that if I had had the opportunity of drawing my conclusions after having heard these papers, I should not make them different. I regret that more was not said regarding the use of vaccines in relation to the nose, and ears. The fact that the climatologists seem to have gotten away with the discussion may perhaps be explained by the well known timidity of the rhinologists and the well recognized modesty of the otologists. One point was brought out by Dr. Birkett and confirmed by the negative reports of results found by both Dr. Cobb and Dr. Coakley, that one positive result is worth many negative findings. Then in his conclusion that treatment by vaccines will sterilize the accessory sinuses of the nose—and there seems to be evidence to back up the statement—he has given us something very important to think about. None of the essayists spoke of many positive results having been obtained in the treatment of the nose—and if that be true, that we can bring about a state of sterility of the secretions, much can be accomplished by the treatment, even though some remains. None of us are afraid of sterile secretions from the nose. We have good reason to be afraid of infectious secretions from the nose.

AUTHORS' INDEX

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OTOLOGICAL LITERATURE FROM JUNE 1, 1909, TO JUNE 1, 1910,

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TRANSACTIONS

OF THE

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FORTY-FOURTH ANNUAL MEETING.

HOTEL CHELSEA,

ATLANTIC CITY, N. J.

JUNE 26 and 27, 1911.

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American Otological Society.

The Forty-fourth Annual Meeting of the American Otological Society was held at the Hotel Chelsea, Atlantic City, N. J., June 26 and 27, 1911.

Members of the American Otological Society, present at the meeting at Atlantic City, June 26 and 27, 1911:

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DRS. FREDERIC E. SONDERN, New York, N. Y.
G. HUDSON MAKUEN, Philadelphia, Pa.
JOHN J. THOMSON, New York, N. Y.

Executive Session, June 26.

Appointment of Business Committee: John B. Rae, New York City, Chairman; F. L. Jack, Boston; Ewing W. Day, Pittsburg, Pa.

Report of Secretary and Treasurer. It was moved and seconded that the report be accepted and placed on file. Carried.

Report of Membership Committee postponed, upon motion, until the afternoon session.

Report of Committee on the Instruction of the Deaf Child. It was moved and seconded that the report be accepted and placed on file. Carried.

Full report of Committee follows the Minutes. See p. 289.

The following were nominated for membership in the Society: Dr. J. J. Thomson, New York City—Proposed by Wendell C. Phillips; seconded by John B. Rae. Dr. Hugh B. Blackwell, New York City—Proposed by Robert Lewis; seconded by William Sohier Bryant. Dr. T. Laurance Saunders, New York City—Proposed by Robert Lewis; seconded by William Sohier Bryant. Dr. O. A. M. McKimmie, Washington, D. C.—Proposed by Charles W. Richardson; seconded by James H. Bryan. Dr. Samuel McCullagh, New

York City—Proposed by T. P. Berens; seconded by J. A. Kenefick. Dr. Antonio P. Voislawsky, New York City—Proposed by T. P. Berens; seconded by John B. Rae. Dr. Alfred M. Amador, Boston, Mass.—Proposed by F. L. Jack; seconded by D. H. Walker. Dr. G. Hudson-Makuen, Philadelphia—Proposed by Charles W. Richardson; seconded by Ewing W. Day. Dr. J. R. Wilson, Chicago, Ill.—Proposed by Norval H. Pierce; seconded by Charles W. Richardson. Dr. Stephen H. Lutz, Brooklyn, N. Y.—Proposed by William C. Braislin; seconded by H. A. Alderton. Dr. F. T. Hopkins—Proposed by Robert Lewis; seconded by F. J. Blodgett. Dr. Charles E. Perkins—Proposed by J. A. Kenefick; seconded by William Sohier Bryant.

The Secretary read a communication from Dr. Samuel Theobald, of Baltimore, concerning the re-numbering of the Transactions. On motion, seconded and carried, the matter was referred to the Publication Committee, with power to act.

Resignations, formerly presented, of the following members were read, and, on motion, seconded and carried, were accepted with regret: Dr. Charles H. May, New York City; Dr. Carl Koller, New York City; Dr. Rudolph O. Born, New York City; Dr. Thomas R. Pooley, New York City.

Reception of guests.

Executive session adjourned.

Executive Session, June 26, 2.30 P. M.

Report of Committee on Admissions: Reported upon favorably, Dr. Max A. Goldstein, St. Louis; Dr. H. P. Mosher, Boston; Dr. H. W. Loeb, St. Louis. Recommended to be carried over until next year, Dr. B. L. Shurley, Detroit, Mich. Recommended for Honorary Membership, Dr. Thomas R. Pooley, New York City.

Moved, seconded, and carried, that the above report be accepted, and placed on file.

Executive session adjourned.

Dr. S. MacCuen Smith, Philadelphia, read a paper on Intracranial Lesions Complicating Acute Aural Disease.

Discussed by Dr. Walter B. Johnson, Paterson; Dr. James F. McKernon, New York; Dr. Henry A. Alderton, Brooklyn; Dr. Norval H. Pierce, Chicago; Dr. Ewing W. Day, Pittsburg; and by Dr. S. MacCuen Smith, Philadelphia.

Dr. Frederic E. Sondern, New York, read a paper on Blood Cultures in Otology.

Discussed by Dr. Robert Lewis, New York; Dr. James F. McKernon, New York; Dr. William K. Rogers, Columbus; and by Dr. Frederic E. Sondern, New York.

Dr. John B. Rae, New York, read a paper on Report of a Case of Sinus Thrombosis with Unusual Metastasis.

Dr. Frank J. Blodgett, New York, read a paper on Report of a Case of Sinus Thrombosis.

Discussed by Dr. B. Alexander Randall, Philadelphia; Dr. Charles W. Richardson, Washington; Dr. James F. McCaw, Watertown; Dr. Henry A. Alderton, Brooklyn; Dr. John B. Rae, New York; and by Dr. Frank J. Blodgett, New York.

Dr. Ernest de Wolfe Wales, Indianapolis, read a paper on Physiology of the Cochlea.

Discussed by Dr. B. Alexander Randall, Philadelphia; and by Dr. Ernest de W. Wales, Indianapolis.

Dr. D. Harold Walker, Boston, reported his failure to finish his experiments on Observations on Intra-labyrinthine Pressure.

Dr. Francis R. Packard, Philadelphia, read a paper on Some Early Books on the Education of the Deaf.

Dr. Charles W. Richardson, Washington, read a paper on Rarefying Osteitis of the Mastoid Portion of the Temporal Bone.

Discussed by Dr. Norval H. Pierce, Chicago; Dr. Ernest de W. Wales, Indianapolis; and by Dr. Charles W. Richardson.

Dr. Philip D. Kerrison, New York, read a paper on Labyrinthine Vertigo.

Discussed by Dr. William C. Braislin, Brooklyn; Dr. B. Alexander Randall, Philadelphia; Dr. Frederick L. Jack,

Boston; Dr. Norval H. Pierce, Chicago; Dr. Joseph A. Kenefick, New York; and by Dr. Philip D. Kerrison.

Dr. B. Alexander Randall, Philadelphia, read a paper on A Note on the More Efficient Methods of Aural Massage.

Discussed by Frederick L. Jack, Boston; Dr. Walter B. Johnson, Paterson; Dr. Sargent F. Snow, Syracuse; Dr. Norval H. Pierce, Chicago; Dr. S. MacCuen Smith, Philadelphia; and by Dr. B. Alexander Randall.

Executive Session, June 27, 10 A. M.

Report of Committee on Officers for next year:

President—Dr. Edward B. Dench, New York City.

Vice President—Dr. Charles W. Richardson, Washington, D. C.

Secretary and Treasurer—Dr. James F. McKernon, New York City.

Committee on Membership—Dr. F. L. Jack, Boston, Dr. Fred Whiting, New York City, Dr. Francis R. Packard, Philadelphia.

Committee on Publication—Dr. B. Alexander Randall, Philadelphia, Dr. Arthur B. Duel, New York City, Secretary, ex-officio.

Committee on Time and Place of Meeting—Dr. Edward B. Dench, New York City, Dr. F. L. Jack, Boston, Dr. James F. McKernon, New York City.

It was moved and seconded that the Secretary be instructed to cast a single ballot in the affirmative for the officers as nominated. Carried. The Secretary declared the ballot cast as ordered, and the officers elected as proposed.

Dr. B. Alexander Randall, of Philadelphia, raised the question of the advisability of arranging the time and place of meeting of the Society to conform to that of the American Ophthalmological Society. After free discussion of the subject the matter was referred, on motion seconded and carried, to the Committee on Time and Place of Meeting, with power to act.

Dr. H. O. Reik, as American Secretary of the International Otological Congress, made a statement with reference to the meeting of the Congress in Boston, June 12 to 17, 1912, and asked the co-operation of the American Otological Society in making the meeting a success.

Dr. B. Alexander Randall moved that a committee be appointed by the Chair to consider the matter of the entertainment by the American Otological Society of the International Congress, such committee to be empowered to draw upon the treasury to the extent of two hundred dollars for such entertainment as they may decide upon. Seconded.

The following amendment to this motion was made: That a contingent assessment of not more than five dollars, in addition to the usual assessment, be made, if necessary. Seconded.

The motion, as amended, was seconded and carried.

Executive session adjourned.

Dr. Norval H. Pierce, Chicago, read a paper (with exhibition of instruments) on A New and Simple Method of Sound Exclusion for the Purpose of Diagnosis in Unilateral Deafness and for Malingering.

Discussed by Dr. B. Alexander Randall, Philadelphia; Dr. Walter B. Johnson, Paterson; Dr. Henry A. Alderton. Brooklyn; Dr. Ernest de W. Wales, Indianapolis; and by Dr. Norval H. Pierce.

Dr. Henry A. Alderton, Brooklyn, read a paper on A Symptom of Mastoid Disease.

Discussed by Dr. S. MacCuen Smith, Philadelphia; Dr. Lewis H. Taylor, Wilkes-Barre; Dr. Ernest de W. Wales, Indianapolis; Dr. Walter B. Johnson, Paterson; Dr. D. Harold Walker, Boston; Dr. B. Alexander Randall, Philadelphia; Dr. Sargent F. Snow, Syracuse; Dr. S. MacCuen Smith, Philadelphia; and by Dr. Henry A. Alderton.

Dr. William Sohier Bryant, New York, read a paper on Reflexes from the Upper Air Tract, Including the Nose and Pharynx.

Discussed by Dr. Joseph A. Kenefick, New York; Dr. B. Alexander Randall, Philadelphia; Dr. Charles W. Richard-

son, Washington; Dr. S. MacCuen Smith, Philadelphia; and by Dr. William Sohier Bryant.

Dr. Norval H. Pierce, Chicago, read a paper on Remarks on the Differential Diagnosis of Serous and Septic Meningitis.

Discussed by Dr. Ewing W. Day, Pittsburg; Dr. Walter B. Johnson, Paterson; Dr. S. MacCuen Smith, Philadelphia; Dr. Edward B. Dench, New York; Dr. James F. McKernon, New York; and by Dr. Norval H. Pierce.

Dr. William C. Braislin, Brooklyn, read a paper on The Sigmoid Sinus and Jugular Bulb of Infants.

Discussed by Dr. B. Alexander Randall, Philadelphia; and by Dr. William C. Braislin.

Dr. Henry A. Alderton, Brooklyn, read a paper on A New Instrument to Remove the Nasal Wall in Cases of Empyema of the Maxillary Antrum.

Discussed by Dr. James F. McCaw, Watertown; and by Dr. Henry A. Alderton.

Dr. Sargent F. Snow, Syracuse, read a paper on The Relation of Active Autointoxication to Acute Middle Ear and Mastoid Inflammations.

Discussed by Dr. Joseph A. Kenefick, New York.

REPORT OF THE COMMITTEE ON THE INSTRUCTION OF THE DEAF CHILD.

COMMITTEE—DR. EDWARD BRADFORD DENCH, Chairman; DR. JAMES F. McKERNON, DR. H. S. BIRKETT.

Gentlemen:—

Your committee begs to report that early in the summer of 1910 a circular letter was sent to 137 Medical Colleges in the United States and Canada, notifying them that a committee had been appointed to consider the better instruction of the deaf child, and requesting each college to have delivered during the senior year, at least one lecture upon this subject.

Out of the 137 letters sent, 92 colleges responded; 45 colleges failed to respond to the circular letter, although this letter was sent, at least, four times during the year.

Of the 92 colleges which responded:

61 Colleges agreed to deliver the lecture.
1 College made a partially favorable report.
10 stated that the matter was referred to the faculty.
3 declined to give the lecture, stating that the course was already too heavy.
5 of the Colleges had been discontinued, and
7 Colleges wrote for further instructions.
5 Colleges stated their course was restricted to the first two years, and, consequently, no instruction of this kind was given.

92

The 7 colleges writing for further information, were as follows:

Maryland Medical College of Baltimore,
Tulane University of New Orleans,
University of Tennessee, at Nashville,
Milwaukee Medical College, at Milwaukee,
Drake University of Des Moines,
Williamette University, at Salem, Oregon, and the
University of Buffalo.

All of the above colleges requested either a Syllabus, so that the lecture could be given along certain lines advised by the committee, or requested that some member of the committee deliver the lecture. As, naturally, the latter request could not be complied with, it seemed wise to the committee to have a regular Syllabus drawn up which could be presented to such institutions requesting information of this character.

A committee having the same object in view was appointed by the American Laryngological, Rhinological and Otological Society, at their last meeting. This committee consisted of your President, Doctor H. S. Birkett, of Montreal, and Doctor G. Hudson Makuen, of Philadelphia. At the last meeting of the American Otological Society, I moved that the same committee be appointed from the American Otological Society. This was done in order to simplify the work. I have discovered lately that Doctor G. Hudson Makuen was not a member of our Society. As Doctor Makuen, however, drew up a model Syllabus for the American Laryngological, Rhinological and Otological Society, I have ventured, as President of the American Otological Society, and after conference with Dr. Birkett, to appoint in his place our Secretary, Doctor James F. McKernon. The committee has decided that it would be wise to embody this Syllabus of Doctor Makuen's in our report.

Respectfully submitted,

E. B. DENCH,
Chairman.

SYLLABUS FOR A LECTURE TO MEDICAL STUDENTS ON THE DEAF CHILD.

Deafness in Childhood.

Deafness the natural condition of early infancy. Hearing an acquired faculty developed during the second, third or fourth months. Refinements of hearing largely a matter of education.

Diagnosis of Deafness.

Positive diagnosis of deafness exceedingly difficult, especially in the first, and sometimes even in the second and third year. Tests for hearing power best made behind the child's back, in order to eliminate the faculty of seeing. Concealed bells and whistles of various degrees of loudness may be used. Muteness oftentimes the first indication of deafness.

Deaf Mutism.

Classification: Congenital. Acquired. Congenital deaf mutism occurs in those born with no potentiality for hearing. Acquired deaf mutism the result of post-natal affections of the organs of hearing, arising from either peripheral or central inflammatory conditions which usually extend to the labyrinth. Deaf mutism acquired after the development of speech results in a class of so-called semi-mutes, or those who have some remnants of automatic speech remaining, although the hearing may be entirely lost. Neglected deafness acquired as late as the sixth, seventh, or even eighth year may result in dumbness.

Diagnosis of Deaf Mutism.

Muteness not pathognomonic of deafness, other possible causes being, a faulty development of the psycho-motor or kinesthetic areas of the brain, and certain forms of paralysis of the peripheral organs of speech due to a faulty development of their motor centers in the bulb and spinal cord. The character of the speech an index to the character of the hearing, defective speech generally denoting defective hearing. Partial deafness may be suggested by the omission of certain sounds of speech. Mutism not necessarily accompanied by absolute deafness. Slight deafness may result in mutism when the mentality is not of high grade.

Physical Characteristics.

Physically, the deaf child differs from the normal child only in respect to the conditions which cause the deafness. The brain of the adult deaf mute, however, may be some-

what smaller than the average brain, and the respiratory organs may show some lack of development on account of their relative inactivity.

Psychical Characteristics.

The mental operations of deaf children who are taught to speak differ in no essential respects from those of hearing children. The natural sign language, or the acquired manual or finger language, not adequate to precision or rapidity of thought, and requiring the use of different parts of the brain. Speech distinguishes man from the lower animals and is absolutely necessary to the highest psychical development. Lower animals have a language of their own, but they cannot speak. Deaf mutes who have not learned to speak are mentally of higher grade than the lower animals because of their inheritance and of their greater powers of expression. During the first year, or prior to the period of beginning speech development, congenital deaf mutes differ but little mentally from normal children. Deaf mutes who have learned to speak and to understand speech by the modern methods may be but little, if any handicapped mentally by their deafness, because the only necessarily undeveloped cerebral area is that employed in audition, the visual area being specially trained to take its place.

Pathology.

Pathology of deaf mutism not well established on account of the difficulties in getting accurate clinical histories combined with adequate postmortem findings. Arrested development of the auditory centers of the brain not easily distinguished. Evidences of pre-existing middle and inner ear diseases frequently found.

Etiology.

Heredity an important factor. The affection may skip one or more generations to appear in a later one. The intermarriage of deaf mutes, and even of hearing persons having a deaf mute ancestry should be discouraged. Labyrinthine

disease, whether originating centrally or peripherally, the most frequent direct cause of deaf mutism. Cerebro-spinal menengitis, measles, scarletina, and typhus fever frequently followed by deafness. Inflammatory conditions of the middle ear, so common in infants and sometimes resulting in extensive destruction or ossification of the contents of the tympanic cavity, are also causes.

Prevention.

Prevention of deaf mutism entirely possible in many instances. Modern advances in Otology not sufficiently utilized in the treatment of very young children. This is a field almost wholly unexplored by the specialist. Inflammatory conditions of the middle ear at birth, the rule rather than the exception, and many children having acquired deafness during the first year are classed as congenital deaf mutes.

The prevention of deaf mutism consists in a vigorous. scientific treatment of the early causes of deafness, and it should, of course, be conducted by the experienced otologist. Such skilled medical attention would probably greatly diminish the number of deaf mutes.

Treatment of Deaf Mutism.

Medical.

Deaf mutism being a condition following deafness of varying degrees and from numerous and diverse causes, requires, first of all, the attention of the skilled otologist. The amelioration of the deafness, and even a cure of the deaf mutism, in the first two or three years among the possibilities. Prompt attention to middle ear disease of all kinds, and all symptoms and diseases referable to the ear. The development of increments and zones of hearing by the use of musical instruments; by tuning forks, as suggested by Bezold; and by the use of the speaking voice in close approximation to the ear, as practised by Urbanschitsch, myself, and others.

Pedagogical.

The education of the deaf mute should begin with early infancy. Training of the special senses. The training and development of latent hearing power especially useful in the production and modulation of the voice. The sense of touch easily developed in childhood and quickly lost if not practised. Kinesthetic areas of the brain of great importance to the deaf in the acquirement of satisfactory phonation and articulation. The training of the visual sense for lip or speech reading also of great importance. The desirability of encouraging the babbling and prattling of deaf infants, and of talking to them as if they could hear. Early neglect and undue sympathy the greatest obstacle to success. All this practicable during early childhood and in a well conducted home. An intelligent mother or governess, with a little instruction from the physician or teacher, may save bright deaf children from the stigma of muteness. Many deaf children found in ill-conducted homes, and hence, the desirability of special institutions for their early education. Differences of opinion as to whether the deaf who have learned to speak should be educated together with or apart from their hearing fellows. The possibility of exceptionally bright children progressing satisfactorily in schools for the hearing, and the impossibility of any but the exceptionally bright accomplishing this task. The teaching of phono-articulation and lip or speech reading to the deaf, a difficult procedure and requiring enthusiasm, patience, knowledge, and skill. Success in the work dependent largely upon the teacher's ability to arouse interest on the part of the patient. Helen Keller's remarkable success due in great measure to her great desire and determination to speak.

Schools for the Deaf in the United States. See the Laryngoscope, December, 1910.

LITERATURE.

The Author's Journal Articles:

The Medico-Educational Problem of the Deaf Child. Read before The American Laryngological, Rhinological and Otological Society, June, 1911.

Development of the Faculty of Speech. International Medical Magazine, July, 1903.

Retarded Development of Speech in Young Children. Pennsylvania Medical Journal, June, 1905.

Relation of the Faculty of Hearing to the Faculty of Speech. The Laryngoscope, January, 1909.

The Physiology and Psychology of Hearing with Special Reference to the Development of Speech. The Laryngoscope, June, 1910.

Teaching the Deaf Child to Hear. New York Medical Journal, March, 1906.

Symposium on the Deaf Child. (Contributed by Physicians and Teachers). The Laryngoscope, June, 1910, and The Volta Review.

Farrar's Historic Introduction to Dixon's Translation of Bonet's "Method of Teaching Deaf Mutes to Speak."

Farrar's Edition of Arnold's "Manual for Teachers on the Education of the Deaf." Book I.

Love and Addison's "Deaf Mutism." Chapters I to VII.

Hartman's "Deaf Mutism."

Farrar's "British Bibliography of the Education of the Deaf." Contained in Vols. I and II of "Teacher of the Deaf."

Elliott's "Articulation and Lip-Reading."

Nitchie's "Self Instructor in Lip-Reading."

Story's "Speech for the Deaf."

Bell's "Principles of Speech."

Kinsey's "Articulation."

Literature and General Information on the Subject Supplied by the Volta Bureau, Washington, D. C.

ADDRESS OF PRESIDENT.

Gentlemen:

It gives me great pleasure to address you on the occasion of the Forty-fourth Annual Meeting of the American Otological Society. The demands upon our time are so urgent, owing to the number of papers to be presented at this meeting, that I shall burden you only with a few introductory remarks.

I first wish to thank you for the great honor which you have conferred upon me in selecting me as your President. The American Otological Society represents all that is best in Otology in our own country, and I cannot tell you how deeply I feel the honor of being your President on this occasion.

The progress in Otology, during the last fifteen years has been so rapid, that that specialty may have been said to have practically been born again. The advances which the otologist has made in major surgery are second to none in any other branch of this science.

During the past year we have lost two of our oldest and most valued members,—Doctor Herman Knapp, of New York City, who died on the first of May, in the 80th year of his age, and Doctor Charles J. Kipp, of Newark, N. J., who passed away on the 13th of January, in the 72nd year of his age.

Dr. Knapp died at Mamaroneck, N. Y., his country home, on May 1st, 1911, at the age of 80 years. He was born in Dauborn, Prussia, on March 17th, 1832. He was educated at the Universities of Munich, Leipsig, Würzburg, Berlin, Zürich, Vienna and Giessen, receiving his medical degree from the last-named university, in 1854. He studied Ophthalmology for four years in the clinics of Paris, London, Utrecht, Berlin and Heidelberg, and in 1864 became Professor of Ophthalmology in the University of Heidel-

berg. While at Heidelberg he founded a hospital and dispensary for eye diseases, which later was incorporated into the university. In 1868, Doctor Knapp came to New York and began the practice of Ophthalmology, which he followed with unusual success, up to the time of his retirement, a few years ago. In 1869 he established the Archives of Ophthalmology and Otology, published in English and German, and the same year he founded the New York Ophthalmic and Aural Institute. In 1882, he became Professor of Ophthalmology in the New York University Medical College, and in 1888, on the death of Dr. Agnew, he was appointed to the same chair, in the College of Physicians and Surgeons. This chair he held until 1902, when he was made Professor Emeritus. Dr. Knapp contributed many articles on diseases of the eye and ear to medical literature, and wrote one complete treatise, on "Intraocular Tumors."

Dr. Charles J. Kipp, of Newark, died at his home, of pneumonia, on January 13th, 1911, aged 72 years.

Dr. Kipp was born in Hanover, Germany, but received his medical education at the College of Physicians and Surgeons, New York, from which he was graduated in 1861. During the Civil War he served first as surgeon to the 5th Reg. N. G., N. Y., and later was assigned to the 3rd Battalion, New York Artillery. In 1864 he was made surgeon of the United States Volunteers, and the following year was brevetted Lieutenant-Colonel. In 1869 he began to practice in Newark, organizing the first eye and ear clinic at St. Michael's Hospital, in 1870. He served as president of the Essex County Medical Society in 1880, and of the New Jersey State Medical Society, in 1886. From 1901 to 1906, he was president of the New Jersey State Tuberculosis Sanatorium, and was president of the American Ophthalmological Society in 1907, of the American Otological Society in 1908, and vice president of the American Medical Association, in 1909. At the time of his death, he was also a member of the Ophthalmic Society of Heidelberg, Germany, of the New York Academy of Medicine and the New York Pathological Society.

He was surgeon to the Newark Eye and Ear Infirmary and consulting surgeon to St. Barnabas, the German, Bayonne, Mountainside and Somerset Hospitals, and chief of the Eye and Ear Infirmary of the Newark City Hospital. His contributions to medical literature were numerous.

Both of these men were pioneers in Otology in this country, and to both of them the younger members of the Society owe a debt of gratitude for the scientific work which they accomplished during their life-time, and for the example which they set us all. Although, at the time of their death, they had passed the allotted span of three-score-years-and-ten, they were always foremost in the field of scientific work, and their lives should afford us an example of what the progressive medical man should be.

Of the progress in Otology, during the past year, it is unnecessary for me to speak. You all of you know of these advances as well as I do, and it is not necessary for me to burden you with an exhaustive history of what has been done in this branch of medicine during the last twelve months.

Of one point, however, I would speak, and that is, the increased attention which has been given to the education of the deaf child. You will remember that at our last meeting, a Committee was appointed to consider the better instruction of the deaf child. A similar Committee was appointed by the American Laryngological, Rhinological and Otological Society and by the Otological Section of the American Medical Association. The Committees of these two former Societies have already reported, and the Report of the Committee appointed by our Society, will be made at a later session. I am pleased to say that very material advances have been made along this line, and I believe within the next few years the medical student will be better able to give proper advice to this class of unfortunates than ever before. It is indeed surprising what can be done with some of these children if they are given a proper course of instruction, and I believe that this Society can do much to improve the condition of these unfortunates.

I would also call the attention of the Society to the fact that the International Otological Congress will be held in Boston, in August of next year, under the Presidency of Doctor Clarence J. Blake. Many of us have been the recipients of courtesy at the hands of our foreign confrères at the various meetings of the International Otological Congresses, held abroad, and it is to be hoped that all of our members will make a special effort to attend the meeting of the International Otological Congress, at Boston, and will do all that they can to accord to our visiting confrères a hearty welcome. Much can be done by individual members in the way of securing a large attendance from abroad, and I would strongly urge that a personal effort be made by members in this Society, to secure as large an attendance as possible upon this occasion. I believe, with all due modesty, that as good work is done in America, in Otology, as is done in Germany, France or England. It is a lamentable fact, however, that this work does not receive the same recognition on the other side of the Atlantic as the work of foreigners receives here. This, I think, is largely due to the fact that Americans travel more, perhaps, than any other nationality, and that, therefore, we have the opportunity of seeing more of the work abroad than our coworkers on the other side of the water have of seeing ours. It is to be hoped, therefore, that on the occasion of this meeting in 1912, the American otologists will make an effort to show their colleagues of other nationalities what is being done in America. We can certainly point with commendable pride to much original work in our country, and with our clinical facilities, there is no reason why we should stand second to any nation as regards our scientific attainments.

With these suggestions, and with a cordial welcome to all, I beg to declare the Forty-fourth Annual Meeting of the American Otological Society opened for the transaction of its regular business.

INTRACRANIAL LESIONS COMPLICATING ACUTE AURAL DISEASE.

By S. MacCUEN SMITH, M. D., Philadelphia, Pa.

Modern pathology no longer limits itself to the restrictions imposed by the terms septicemia and pyemia. Such terms belong to a period antedating our conception of modern advanced bacteriology. This science has demonstrated that surgical sepsis and accidental wound infection are not the only etiologic factors, but that there is a variety of pyogenic infections originating in obscure processes and from unknown causes, to which the term "cryptogenetic" (Leube) has been applied.

Septicemia has been designated as a condition in which pyogenic organisms disseminate from a primary focus into the general blood supply, settle, collect, and multiply in the capillaries. In pyemia, they are disseminated, the metastatic suppuration originating in a primary focus. As a matter of fact, both of these conditions actually co-exist.

Clinically, a strict differentiation between these two conditions is impossible from a purely scientific standpoint, as the number and virulence of the micro-organisms which find their way into the circulation will at one time give rise to localized metastases and at another to general septicemia. As a matter of fact, transitional forms arise (and these are the most common) and we then refer to septico-pyemia or, briefly, "sepsis".

Of great importance for the proper understanding of the clinical condition "sepsis" is the knowledge that in different cases various organs and regions of the body may offer the principal focus for the attack of the bacteria. This gives rise to serious local disease, which naturally stamps the

affection with a certain degree of individuality and accounts for the fact that such diseases were formerly regarded as special maladies. While clinically there may be the greatest difference between the septic infection of a cerebral sinus and a so-called malignant endocarditis, pathologically they are both mere manifestations of sepsis with a local point of selection.

As is well recognized, the chronic forms of aural inflammation frequently cause intracranial lesions. A consideration of the pathologic changes wrought by the micro-organisms of a chronic discharge enables us to appreciate how these purulent products, through osseous softening and carious erosion, produce intra-venous and intracranial complications. Although in the past much stress has been laid on the development of endocranial lesions as a complication of the **chronic** forms of aural disease, we must not lose sight of the fact that these grave diseases probably arise more frequently from the **acute** infections than we have formerly suspected. This is especially true in young subjects, due, no doubt, to the fact that the very thin and porous osseous walls offer but an ineffectual barrier to the ingress of infectious micro-organisms, thereby actually favoring a primary thrombus of the bulb in the one instance, and meningitis in the other.

These infectious processes may arise either through the numerous venous connections or by means of their intimate juxtaposition. When arising from the acute form of disease, the infection is carried by means of minute thrombi through the mucosal or osseous veins, into the sinus wall, forming a mural thrombus, which may continue to increase in size until the vein is entirely occluded. In the chronic form of aural inflammation, an endocranial complication usually arises primarily from osseous softening and subsequent carious erosion, the result of contiguity. When the sigmoid sinus becomes infected, the thrombi may extend, by way of the internal jugular, to the superior vena cava, which was demonstrated in the postmortem findings in one of my cases; or they may involve the cavernous sinus and ophthal-

mic vein by extension anteriorly through the superior or inferior petrosal sinus, or the thrombi may travel posteriorly to the torcular and superior longitudinal sinus, the resulting septic thrombus being discovered at the autopsy, although the condition was not suspected during the ante-mortem state.

It is claimed that septic thrombi may be carried to the interior of the brain by the backward motion of the blood current when the lateral sinus is obstructed or obliterated, giving rise to metastatic abscess formations. This may explain the method of infection in three cases of hepatic abscess by aural metastasis, reported by me in 1904. undoubtedly the back current, or regurgitation from an obstructed sinus on one side is capable of producing a similar condition on the opposite side through the torcular.

The clinical symptoms of septic sinus thrombosis indicate that the onset is always sudden; although actual involvement of the sinus itself is a progressive process, usually very gradual, even when arising from acute aural disease. This is well illustrated in the report of a case incorporated in this paper. The presence of a sterile clot accounts for the absence of more or less characteristic symptoms in the early stages, but it later becomes septic through bacterial invasion, softens, and disintegrates. However, a septic thrombus may wholly disappear through the process of absorption, resulting in complete obliteration of the sinus, which, through thickening and contraction of the walls, is converted into a hard, fibrous-like cord. Such conditions are not infrequently found at autopsies, and would seem to explain or deny the implied theory of their congenital origin.

On the other hand, a septic thrombus, through disintegration, may be converted into free pus and remain inactive so long as both ends are securely sealed by a sterile plug and the vessel wall remains intact. The case herein reported illustrates this point.

Since the memorable operation in 1886, by Barker, of London, who successfully evacuated an abscess formation from the temporal lobe, in which case the patient presented no

external signs whatever of his malady, we have been laboring, and not in vain, for the betterment of our surgical technic, and the development of more accurate methods in brain localization. In the first instance our success has been notable; in the latter, disappointment and chagrin still confront us in a goodly number of cases, and yet, even in brain localization, the greatest problem of medical science today, we can report progress. It seems, therefore, that the dawn of a new era is upon us, and that in the near future we will have mastered this perplexing problem. The pathology of brain abscess formation is identical with that of sinus thrombosis; their occurrence, would, no doubt, be much more frequent were it not for the obstacles offered by the dura in its resistance to microbic invasion. In this connection, we must bear in mind the important fact that the dura will more readily succomb to sudden or accidental infectious exposure than to the usual slow process by carious erosion. In the latter, the firm resistance of the strong fibrous tissue comprising the dura is greatly increased by considerable thickening, and later still further fortified and protected by the formation of granulation tissue.

By means of the blood stream and perivascular sheaths, pathogenic micro-organisms are frequently carried to the interior of the skull, causing brain abscess formations, even though there may be no recognizable softening or carious erosion adjacent to the site of local infection in the tegmen antri or tegmen tympani; this etiologic factor is true, also, in the development of phlebitis and sinus thrombosis, but I believe to a much less extent. On the other hand, as a medium of transmission for the production of meningitis and the various metastases throughout the economy, the blood and lymph route is by far the more common.

The following cases, one of sinus thrombosis and the other of brain abscess, are interesting and somewhat illustrative of the few points in pathology mentioned above.

Case I. Patient, A. D., female, aged 27 years. Admitted to the Germantown Hospital, January 28, 1911. She suf-

fered from severe pain in the right ear for about one week. Claimed never to have had an aural lesion of any description prior to the present illness, and this statement was substantiated by members of her family. Three days prior to admission to the hospital, she suddenly developed a Bell's palsy, noticing on awakening in the morning that she could not completely close her right eye nor freely move the right side of her face. At the same time she noticed that her speech was thick.

The patient had an anxious expression, her skin was hot and quite moist, and the pulse soft and rapid. There was no tenderness or other abnormal symptom noted over the mastoid region, and examination of the external auditory canal showed nothing except a mass of detritus. This could be removed only in part, as the slightest manipulation caused considerable pain. The right side of the neck was swollen from the region of the mastoid almost to the clavicle. This swelling was slightly red, quite tender, and entirely free from fluctuation.

The above-mentioned plug of inflammatory debris was removed from the auditory canal on the following day, revealing the destruction of the greater part of the drumhead. Temperature on this date was 102.4° and pulse 120.

A mastoid operation was performed, on February 3, under general anesthesia. On dividing the periosteum, the bone bled freely, there being but little change otherwise in the cortex. After removing the cortex, the mastoid cells were found to be soft and necrotic, containing considerable inspissated pus. The tip, however, was not involved, and there was no exposure of the sinus, the osseous covering of which appeared not to have undergone any pathologic changes.

Although I anticipated performing only a simple mastoid operation, the necrotic changes involving the posterior canal were so great that it was necessary to do a complete operation. Assuming that the swelling in the neck represented a Bezold's variety of mastoid disease, a grooved director was introduced into this swelling at the lower angle of the pri-

mary incision, but no pus was evacuated. I felt, therefore, that this was an enlarged gland.

With the exception of a rise to 104.2° on February 7th, four days after the operation, the temperature gradually assumed a normal course until February 11, when it began to rise. On February 13 the glandular swelling of the neck had diminished somewhat, but there was still considerable tenderness. The patient appeared quite septic.

On this date Dr. Ross, one of the general surgeons of the hospital, operated on the neck, with a view of removing the enlarged gland. An incision in the line of the swelling revealed a large lymph node over the middle portion of the internal jugular, which proved to be responsible for a considerable part of the edema. During the removal of this node, the internal jugular, to which it was firmly adherent, was torn, which was immediately followed by the free escape of considerable pus. After evacuation of this pus, the cavity was packed with iodoform gauze, following which the incision was partially closed with silkworm-gut sutures.

Ten days later the patient was still running a septic temperature. An X-ray showed a distinct shadow in the region of the lateral sinus. A third operation was deemed advisable, and the retro-pinnal incision was re-opened and the lateral sinus exposed. It was about normal in tension and color, and when opened was found to contain thick, ropy pus. There was practically no bleeding until the sinus had been curetted for a considerable distance toward the trochlea. Water was forced through the jugular foramen from above until it flowed from the wound in the neck. The sinus was packed with iodoform gauze, the incision partially closed, and the wound dressed with sterile gauze.

A bacteriological examination of the pus showed pure streptococcic infection.

After this operation the patient was pale and the hemoglobin and red blood count low. She was placed on abundant feeding and was given daily a hypodermic injection of one ampoule of "Injectio ferri compositus" (Citrate of Iron,

gr. j), under which treatment the hemoglobin and red blood count rapidly increased to approximately normal.

On March 1st the pulse and temperature reached normal.

On March 13th an abscess over the right buttock was opened, containing approximately half a pint of pus. The patient stated that she had had this swelling for three weeks, but did not mention it through fear of necessitating further operative procedures.

On March 23d, under nitrous oxid anesthesia, Dr. Ross opened the abscess over the right buttock in three places, and inserted rubber drainage. At the same time a slight plastic operation was performed to close the opening back of the right ear.

On March 30th a small abscess about the middle of the outer side of the left hip was evacuated.

The above case is here reported with a view of showing the great destruction that may take place as a complication of an acute aural infection. It seemed so improbable that within a period of about ten days the carious erosion of the mastoid and middle ear should have been so great, together with the Bell's palsy and lateral sinus and jugular thrombosis, with subsequent disintegration of the clot, as well as metastatic abscess formations, that I made every effort to confirm or disprove the statement of the patient and her relatives that her aural condition was really of the acute variety, and although I was finally compelled to accept their version of the situation, I nevertheless feel somewhat skeptical as to its absolute accuracy. If, however, their statements are true, and of this there seems to be no reasonable doubt, the case is both interesting and instructive to show the rapid destruction and complications arising from this bacterial invasion in an acute aural disease.

Case II. I am indebted to Dr. H. A. Rothrock, of West Chester, Pa., for the detailed history of the following case:

Patient, Dr. J. R. H., aged 30 years. Family history negative. The patient suffered from most of the usual diseases of childhood, and in addition to these, he had typhoid fever

when two years old. About ten years ago he was treated for an acute otitis media, from which he made a prompt and permanent recovery.

On February 25, 1911, he had an attack of "la grippe", but owing to the serious illness of his father, also a physician, he remained in bed but two days, and then attempted to attend to both his own and his father's practice. During this time his temperature ranged as high as 103°. After about three days he became so prostrated that he was again forced to give up work and confine himself to his room. His ear now started to pain him severely, and after two or three days the drum ruptured spontaneously, with a very free discharge. When I first saw him he was still suffering intensely, the discharge was free, of a ropy consistency, and light yellow in color. The entire surface of the deep canal was swollen, obstructing a view of the membrana tympani. His temperature at this time was about 102°, and he seemed to be suffering from the great depression that was characteristic of the whole course of his illness, in marked contrast to his usual happy disposition. After thorough cleansing, aristol was insufflated, and this treatment was continued until the time of the first operation. His diet was restricted and a calomel purge given. Under this line of treatment the discharge abated, the funicular appearance of the canal disappeared, and in two or three days the drum was clearly visible, showing a medium-sized opening in the lower anterior quadrant. Improvement was gradual but steady for a period of about ten days.

On the afternoon of March 12th his condition suddenly changed for the worse. The pain again increased and headache set in. During the night he commenced vomiting, of the projectile type. When seen Monday morning, his temperature was ranging around 104°, the pulse rate 90, and respiration about normal. The tongue was heavily coated, and he complained bitterly of pain in the head, mostly frontal, to which location the headache was referred throughout the subsequent course of the disease. There was no bulging of the postero-superior wall of the canal, no

swelling back of the ear, and pressure failed to show any spot of tenderness over the mastoid region. Vomiting continued more or less during the day, and the patient was still irritable and depressed.

About noon he was admitted to the Chester County Hospital, and Dr. S. MacCuen Smith was called in consultation, seeing him about 8 p. m. By this time the postero- superior wall of the canal was sagging, and deep, hard pressure over the mastoid region elicited some tenderness. Temperature was 104°, pulse rate 90 and respiration 24. Dr. Smith advised and performed a simple mastoid operation. Some granulation tissue and considerable free pus was removed from the lower cells and antrum, securing the establishment of a through-and-through drainage. There was no exposure of either the sinus or dura, the osseous walls covering the same being normal in appearance.

Following the operation, the temperature gradually subsided, the discharge decreased considerably in amount, and with the exception of the stitches becoming infected, the course of the disease was apparently normal. However, the patient's mental depression continued, though even in this there was daily improvement. By March 18th his mentality had greatly improved, and the temperature, pulse and respiration had reached normal. which condition remained until the 21st of March.

On the afternoon of this day the temperature rose to 102°, but promptly dropped again with the evacuation of pus from a stitch abscess in the upper angle of the wound. After this the temperature remained about normal until March 25th, when it reached 100°. The next night the patient was seized with violent headache, which lasted with greater or less severity until within a few hours of the second operation. From this time on the prostration and depression increased daily, and for brief intervals the patient would become delirious, being, however, easily recalled to his surroundings. Mental activity decreased gradually. On the 27th his temperature rose to 100.8°, falling the next day, on which the highest record was 99.4°, and reaching normal

again on the 29th of March for a brief period, after which it rose to 100.4°. During this time the pulse and respiration continued about normal.

On March 31st the temperature was nearly normal at midnight, with a pulse of 56. Again it ascended to 101°, around which point it stayed for the next few days. By this time the delirium was becoming more pronounced, although the patient could still talk and think intelligently most of the time. At intervals there was some apparent weakness of the left side, though not as yet sufficiently marked to be at all positive. The pupils were equal, reacted well, and the eyeground, save for some congestion of the retinal veins, was normal. On this day Dr. Smith again saw the patient, in connection with Dr. John H. Gibbon and Dr. George E. Price. By this time pressure symptoms, probably from a brain abscess, seemed to be definitely marked, but in the absence of localizing symptoms, it was deemed best to wait a day longer before operating, hoping by that time to gain some definite guide as to accurate localization. On April 1st, left arm and leg paralysis was quite well defined, speech was thick, and the mental condition aggravated. Arrangements were made to perform the complete mastoid operation, with exploration of the brain, on the following day. That night the patient's condition became seriously worse, and by morning he had sunk into a typhoid state, from which he aroused for one or two momentary periods, during which he seemed to recognize those around him. There were constant convulsive movements of the right arm and leg, the left side being apparently paralyzed most of the time, although, strangely enough, on two or three occasions he regained their use, showing his remarkable vitality. At 11 o'clock on Sunday morning, Dr. Smith, assisted by myself and Dr. A. Spencer Kaufman, performed a radical mastoid operation. There was not the slightest evidence of further carious erosion in any part of the mastoid or tympanic cavity. The brain cavity was opened through the tegmen antri. The bulging dura showed abnormal intracranial pressure, though no pathologic change was

noticed in the dura itself. On incising the dura, healthy brain matter crowded into the wound. The Jackson brain forceps were introduced, first upwards, inwards and forwards, then upwards and inwards, and again upwards, inwards and backwards. The third time there was a gush of about one ounce of pus from a cavity located about two inches deep from the dural surface. The lateral sinus had already been exposed and found to be healthy. A drainage tube was introduced into the abscess and the wound packed and dressed.

The next day the patient's mental condition was considerably better, but the temperature was now ranging about 104°, the pulse from 120 to 150 and the respiration had reached 32.

On the following day the patient's condition was less satisfactory, and Dr. Smith removed the tube, which was somewhat occluded. This provided for the free escape of some pent-up pus, and resulted in a temporary betterment of the patient's condition. A drainage tube was re-inserted in the old tract.

An examination of the pus showed a pure streptococcic infection. In the meantime autogenous vaccines had been administered. A few days later the pus, which was gradually becoming less in quantity, ceased. The tube was removed, but no pus evacuated. Apparently the abscess had been completely drained.

Despite all that was done, however, the patient's condition became steadily worse and he died on April 6th. The temperature taken in the axilla shortly before death was 106.2°, pulse 180, and respiration 60 to 70. An autopsy was refused.

The differential blood count, as made by Dr. Wellington W. Woodward, is interesting, as it well demonstrates not only the severity of the infection, but the patient's great power of resistance. On the day of the first operation he presented a leucocytosis of 12,000, which gradually increased up to the time of the second operation to 15,800, with a polynuclear percentage of 86.5, whereas the day before his death

the leucocytosis was 22,600 and the polynuclear percentage 93.1.

From the above history it would seem that a brain abscess formed within two weeks of the time of this patient's initial ear lesion. Although he seemed to improve materially after the simple mastoid operation, yet the mental state was never entirely satisfactory, and I feel, therefore, that the collection of pus within the interior of the skull must have been deposited through the medium of the blood and lymph streams very early in the course of the disease, and was present, or in process of formation, at the time of the first operation.

Another point of interest is the fact that I succeeded, apparently, in evacuating the pus from the parent abscess formation. The patient's death, therefore, was probably due to a diffuse meningitis or general encephalitis as a result of ruptured satellite abscesses.

It is also interesting to note that the abscess was situated about two inches beyond the dura, with healthy brain tissue separating these two points.

The refusal of an autopsy denied us the opportunity of ascertaining valuable clinical facts.

Discussion.

Dr. Walter B. Johnson, of Paterson, N. J., said the difficulty, in the first case, was to determine whether or not the condition was due to an acute infection. It was very uncommon for the drum membrane to be entirely destroyed, with a considerable deposit of detritus, after a week's infection of an acute character. In the second case it came after a considerable period of time, and after the mastoid operation, and of course there might have been a primary infection of the brain. The difficulty which presented itself in these cases was the absence of symptoms. Otologists were looking all the time for evidences by which they can localize the presence of abscess. He had come to the conclusion that it is undesirable to wait too long. If there is

evidence that the disease has extended from the ear to the cerebral cavity, an exploratory operative procedure would be permissible. The cases which die as the result of brain abscess prove fatal because otologists are not able to persuade themselves to do a major operation early enough. He did not desire to agitate the question of early operation, but he believed many cases could be saved if operated upon earlier, after all efforts at diagnosis have been made and a reasonably definite conclusion reached. He had recently heard Dr. Martland of Newark, read a paper in which the latter compared the clinical diagnosis with the pathologic anatomic diagnosis in a series of 500 cases, and the clinical diagnosis seemed frequently to be at fault. It was interesting to note that death had probably resulted from failure to institute operative procedure in a considerable number of the cases especially those in which there was cerebral involvement by extension.

Dr. James F. McKernon, of New York City, asked the reader of the paper if the pus evacuated from the second case had any odor. The question being answered in the affirmative, the speaker continued that he did not believe the condition, in that event, resulted from an acute infection. The suddenness of the onset in sinus thrombosis depended upon the virulence of the infection and the patient's resistance. He had seen several cases recently in which the infection was of a low order, and the symptoms presented were entirely different from those ordinarily encountered.

Dr. Henry A. Alderton, of Brooklyn, N. Y., was interested in the case of facial paralysis. In this connection he cited a case in which facial paralysis followed a grippe infection. Otitis media developed, the drum membrane was incised, and he decided to await developments for twenty-four hours. The next morning the doctor in charge said the patient showed loss of power on the opposite side. She was sent to the hospital for operation. The mastoid was found broken

down, with thick pus, extending extra-durably over the petrous portion. The patient died the next morning of leptomeningitis.

Dr. Norval H. Pierce of Chicago, believed there was reason for regarding the first case as chronic, because, as had been said, one rarely sees destruction of the tympanic membrane except in cases of virulent infection, or where the patient is suffering from cachexia. Destruction of the membrane to any considerable extent signified to him that the condition is chronic.

Localization of brain abscess was very important. He cited the case of a little girl who had a swelling over the right mastoid and a scar from a previous operation. She complained of headache. There was no fluctuation in the mastoid swelling, which was rather hard, so that the bone could be very readily felt under the skin, showing that there was no special infiltration of the soft tissues. She was exceedingly nervous and hysterical. Two days after admission there was loss of motion of the opposite side in the face, leg and arm, with convulsive seizures beginning in those members. Her temperature was 102°, the eye grounds showed choked disc, and she was operated upon late that night. He found the bone in the mastoid intact except at the extreme upper portion, where a slight defect was present from the previous operation. The cortex was removed. The diseased portion occupied the lower part and extended backwards, so that in scraping the contents of this cavity away the sigmoid sinus was exposed. A button of bone was removed from the squamous portion of the temporal bone, and the meninges were found to be absolutely normal. In abscesses as large as this there might be pressure on the internal capsule, which could produce paralysis of the opposite side. Whether it was due absolutely to the pressure of the abscess or to the edema which surrounds such abscess had not been decided, but the clinical fact remained. He could have explored the temporal lobe backwards and downwards, but he ceased operating rather than explore the brain for

an abcess which was not well localized. The paralysis increased somewhat after operation, but the patient subsequently made a complete recovery. In this case there was hemiplegia, beginning optic neuritis and localized areas of anesthesia scattered all over the body, evidently hysterical in nature, in a girl twelve years of age. If he had explored the brain the picture would have been changed completely. He believed it best not to make brain explorations unless there are pretty clearly defined reasons for doing so, because the operation is not without danger; on the other hand, if one does not use haste one may be responsible for some deaths. He believed, however, that more deaths would result if exploration is resorted to without adequate reason.

Dr. Ewing W. Day, of Pittsburg, Pa., said he makes a two-step operation in the class of cases under discussion, doing first a decompression operation to relieve the pressure and to ward off danger. If the pus is under pressure it will follow the line of least resistance, which is toward the line of decompression. The abscess is opened the day after the decompression. If one waits twenty-four to forty-eight hours after the decompression operation before probing for the brain abscess the patient is more apt to recover than when one goes right ahead with the complete operation.

Dr. Pierce added that there are two kinds of abscesses, one, connected by a stalk with the original focus of infection; the other, isolated abscesses, to follow up the channels of infection of which would necessitate going through healthy brain substance and meningeal coverings. In the latter class the patients die in the vast majority of instances. Scarcely three per cent. of cases recover where the exploration goes through healthy brain tissue.

Dr. Smith, in closing the discussion, said Dr. McKernon must have misunderstood what he said in the paper about the cases being of sudden onset. His statement was that they appeared to develop suddenly, but in his judgment

they always develop slowly, notwithstanding an irregular or even absent symptomatology during the early stage. Referring to Dr. McKernon's question about the odor, he said he was not sure about it, but would look into the matter. He reiterated what he had said at the end of his paper, that such destruction rarely comes from an acute infection. He had made every effort, by quizzing the patient and the patient's family, to ascertain whether she had had any previous trouble, and had been assured by them that she had had no ear trouble until the present attack.

The question of localization was one of the problems of medicine. In the case cited the general physician and the neurologist were taken to the patient's bedside in order to determine the locality of the brain abscess. The pain complained of was in the frontal lobe, and the supposition was that the abscess was in that region, but in this, and in three other cases of the same character, the pus was found in the temporal lobe.

BLOOD CULTURES IN OTOLOGY.

By FREDERIC E. SONDERN, M. D.

The relative frequency of instances is noteworthy in which the positive or negative result of a blood culture is of distinct diagnostic value to the otologist. The comparatively few published accounts of its use would seem to indicate that this valuable diagnostic aid has not had the general attention in the routine practice of the specialist it deserves.

Emanuel Libman of the Mt. Sinai Hospital is certainly entitled to the credit of having done much original work in blood culture studies in diseases of the ear, and his pioneer publications on this subject are both interesting and instructive. The attempt to base positive and unexceptionable rules solely on the outcome of this procedure is an error just as in any other laboratory aid, and it is this effort that has led to controversy and more or less discredit. The object of this communication is to rehearse briefly the value found in positive as well as negative blood cultures in otological practice and to demonstrate the relative ease of the technical procedure.

In cases of acute purulent otitis media without or with mastoid involvement, bacteria are not usually found in the blood cultures. There are exceptions however, and these cannot always be ascribed to errors in technic as they have occurred in the experience of numerous competent clinicians and accurate laboratory workers. It also does not seem justified to claim that an unrecognized thrombosis exists in these exceptional cases. Fortunately these exceptions are rare particularly after efficient surgical treatment of middle ear and mastoid.

In the cases in which there is a thrombosis of the sinus, bulb or vein, bacteria are found in the blood cultures in a

very large percentage of the cases. Exceptions to this rule also occur. A thrombus is quite firm at first and if it contains but few organisms those may be killed by the bactericidal power of the blood itself. Even if infected to a greater extent, the firm clot may prevent the organisms from getting into the blood current. It is even possible that pieces of infected clot may break off and be carried by the blood to distant parts, causing metastatic suppurating foci without bacterial infection of the blood stream itself. In the large majority of instances however, the softening of the thrombus causes bacteria to be fed to the blood stream in increasing numbers and then positive blood cultures are invariably obtained. Not only an extension of the infection to the sinus with a resulting thrombosis will produce a bacterial infection of the blood current, but a secondary meningitis, a brain abscess or even an extradural suppurative focus may subsequently do the same. It is also very important to remember that these general invasions of the blood current may result from local processes other than in the ear, and the mere fact that a bacterial infection of the blood stream is found naturally does not necessarily indicates otitic origin.

In a given case of acute suppuration of the middle ear with mastoid involvement a positive blood culture should not be accepted alone as certain evidence of a co-existing thrombosis of the sinus. It has been demonstrated as previously stated that exceptionally a bacteriemia does occur without a thrombosis of the sinus, but this is certainly unusual to say the least. After proper surgical treatment of the middle ear and mastoid in these exceptional cases, the bacteriemia should decrease and disappear together with the proper behavior of the condition clinically. On the other hand, should the colonies increase in number in the subsequent cultures, it may be accepted as strong presumptive evidence that an extension of the invasion has occurred. The most frequent extension is into the sinus with resulting thrombosis and for this reason the bacteriemia is usually quite properly looked upon as one of the evidences that such extension has occurred. In the great majority of instances

however, a bacterial invasion of the blood current does not occur in the cases of middle ear and mastoid infection unless there has been an extension into the sinus or elsewhere.

While the presence and particularly an increasing degree of bacteriemia may justly be accepted as an indication of such extension the clinical evidences are imperative to determine what structures have been invaded. It is self understood that the direct invasion of the circulatory apparatus will lead to positive blood cultures far more easily than an invasion of the meninges or brain tissue, and subsequent infection of the blood stream, but after all the clinical symptoms and not the blood culture must determine the nature of this extension. An extension of the process beyond the mastoid may occur and still may not give rise to a bacteriemia for the reasons previously stated, namely, a firm clot with a small or large number of organisms may not cause infection of the blood current until softening of the thrombus occurs. For this very reason it is evident that a single culture if negative may be misleading. When suggestive symptoms occur, a negative blood culture may be used as an argument against sinus thrombosis possibly for the moment, but if these symptoms continue, repeated cultures are necessary as the former negative culture can have no weight whatever.

It is natural that intercurrent diseases or complications of the original causative disease, with consequent elevation of temperature, may occur during the course of an otitic infection. The blood culture aids in the differential diagnosis though careful clinical observation with indicated laboratory aids are at least equally important and sometimes more so. Acute articular rheumatism shows a negative blood culture while a pyemic joint is usually associated with a bacteriemia. Pneumonia usually gives physical signs and possibly a bacteriemia of other type than the otitic infection. Typhoid shows a leucopenia and relative lymphocytosis, and possibly characteristic organisms on blood culture or a positive Widal reaction, while an extension of the otitic infection shows a leucocytosis and relative polynucleosis and

possibly a positive blood culture of the organism found in the ear. Tuberculosis also shows a leucopenia and relative lymphocytosis and even a positive Diazo reaction at times but the blood cultures are sterile and a von Pirquet or Calmette may be positive. Unusual temperatures and disturbing symptoms such as headache and local neuralgia, due to an unrecognized acute nephritis may develop during the convalescence of a scarlet fever mastoid. If perchance a streptococcemia exists, which is not unknown in this disease, an erroneous diagnosis of sinus thrombosis would be easily made if the rule is accepted that a bacteriemia invariably indicates this complication.

The following conclusions seem justified: Early positive blood cultures with an unopened mastoid infection usually indicate co-existing sinus thrombosis. The possible exception must be kept in mind especially in absence of all clinical signs. Negative blood cultures under like conditions usually allow the exclusion of a sinus thrombosis. The possible exception must be remembered especially if suspicious clinical signs exist.

Late positive blood cultures in operated cases of mastoiditis with exacerbations of temperature after a longer or shorter afebrile period, also usually indicate thrombosis. But it is in these cases that particular care must be exercised in concluding that the infection of the blood stream is due to a thrombosis and not to a meningitis or brain abscess, to say nothing of a more distant point of invasion not necessarily secondary to the mastoid suppuration. Negative blood cultures under like conditions usually allow exclusion of a thrombosis and indicate careful search for other causes to explain the temperature curve; but the exceptions to this rule must not be forgotten.

With reference to the organisms found, the streptococcus is without doubt the most frequent invader of the mastoid and deeper structures and in consequence is the organism most frequently obtained in blood cultures. The streptococcus mucosus and the staphylococcus occur next in the order of frequency. While most of us have not seen a sinus

thrombosis follow a pneumococcus infection of the ear the evidence is not yet nearly sufficient to justify a conclusion that it does not occur. With due regard to the difficulty in differentiating streptococci and pneumococci and admitting the inability to do so in some instances, there does seem sufficient evidence at hand to believe that the pneumococcus does act as a pyogenic organism in the mastoid, and consequently it seems difficult to understand that it cannot produce a sinus thrombosis, as well.

It is certainly true that the staphylococcus and pneumococcus found in otitis media are far less apt to cause mastoid and deeper seated complications than the streptococcus and streptococcus mucosus, but absolute rules should not be made concerning the relative virulence of one organism as compared with another or that certain structures are immune to certain bacterial invasions. More searching investigations of the bacteria concerned in the production of the suppurative processes under consideration may teach much of value for the anaerobic organisms present have had but little attention as yet.

Blood culture work in this, as in other fields, should be done quantitatively, that is, not only the organisms should be determined, but the number of colonies per cubic centimetre should be estimated. This is of particular value when cultures are repeated, as a decrease or an increase in the bacteriemia is thus learned. If the organisms present produce hemolysis is a point which should also be carefully observed, as it invariably indicates a more serious condition than when this solution does not occur.

Blood cultures should, when possible, be made by those thoroughly familiar with the laboratory technic, as the distribution of the blood in the different media must be done before coagulation occurs, and to do this accurately and quickly with blood at the point of coagulation requires dexterity only acquired by practice. A suitable vein, if possible, at the bend of the elbow is selected and a constricting bandage is applied above. The region is thoroughly cleansed with green soap, ether and alcohol and a drop of

tincture if iodine allowed to dry where the puncture is to be made. If a properly sterilized 10 c.c. all glass syringe with fine iridoplatinum needle is used in place of the 25 c.c. hospital affair with a trocar-like weapon attached, the patient usually makes no objection whatever to the original or repeated aspiration. Having obtained from 5 to 10 c.c. of blood, an assistant or nurse removes the bandage, holds the arm up for a few minutes and applies a drop of collodion over the puncture. The obtained specimen is quickly and accurately distributed in the different media, namely flasks of plain bouillon and glucose bouillon and plates of glucose agar, serum agar and glucose serum agar. These are incubated at 37.5° C. and examined at 18-hour intervals. The rapidity of growth varies decidedly with the same type of organism and prolonged observation is essential before the culture can be considered sterile.

The difficulty of transporting the various culture media to the bedside in private practice and the danger of partial coagulation before the blood has been accurately distributed, induced Epstein, (Amer. Journ. Med. Sciences, September, 1907) to suggest the use of a method to prevent the coagulation of the blood and thus allow the distribution to be made at leisure in the laboratory.

A large tube containing 10 c.c. of an isotonic ammonium oxalate solution, the whole properly sterilized, is carried to the bedside, into which the aspirated blood is immediately introduced and a complete mixture made. This specimen can then be accurately distributed in the various media at the laboratory. Ryttenberg (Journ. Med. Research XX, No. 1) has demonstrated the comparative value of this method and a limited experience with it tends to corroborate his results. Another advantage it has is that it can be put into the hands of the surgeon for the purpose of obtaining the specimen which is subsequently handled by the laboratory worker.

Discussion.

Dr. Robert Lewis of New York City, cited a case which illustrated one of the points spoken of by Dr. Sondern, viz.:—the absence of a bacteremia in some cases of sigmoid sinus thrombosis.

The patient, a large, robust laborer, was operated upon for a very extensive mastoiditis by Dr. Lewis at the New York Eye and Ear Infirmary, in February last.

The sigmoid sinus from the knee to within a short distance of the bulb was covered with granulations and bathed in pus. Following the complete mastoid operation the patient progressed very favorably for seven days, the temperature gradually declining from day to day until it reached 99.5° F. At eight P. M. on the close of the eighth day following the operation it suddenly rose to 103.2° F., and at eight A. M. the following day the temperature was 105° F., and by four P. M. it was 105.6° F. A blood culture was made at one P. M. on this day; it proved to be **negative at the end of seventy-two hours.** The blood count was: Red blood corpuscles 5,200,000, white blood corpuscles 8,400, small mononuclear 12.2, polynuclear 88.8. A second culture made on the tenth day **was negative at the end of seventy-two hours.** The temperature still remaining about 105° F. on the afternoon of the ninth day and no symptoms of any other complication being present, Dr. Lewis determined to explore the sigmoid sinus. On incising the sinus a solid clot was found entirely filling the lumen of the vessel. The mastoid cavity was temporarily packed with gauze and the operation for ligation and excision of the internal jugular vein was performed. The vein was resected for almost its entire length, and found to be thrombosed throughout. After the neck wound was partially dressed the clot was removed from the sigmoid sinus. On cultivation these clots showed a few long chains of streptococcus pyogenous. Following the operation the temperature very gradually declined to 102° F. the following day, but arose to almost 105° F. on the twelfth day, and then gradually

declined from day to day to 100° F. on the sixteenth day. On the seventeenth day it gradually arose to 104.4° F. and then gradually fell to below 100° F., and was practically normal for some days previous to the discharge of the patient from the hospital on the thirtieth day, at which time the wounds were almost healed.

A bacteremia was not present in this case of complete thrombosis of sinus and vein, so the diagnosis had to be made upon the only symptom present, viz.:—a sudden rise of temperature followed by remission on two occasions within twenty-four hours, accompanying a very extensive mastoiditis.

Dr. James F. McKernon, of New York City, said he was glad to hear Dr. Sondern emphasize the importance of making repeated cultures. He cited a case in which the first three cultures, examined at eighteen-hour intervals, were negative. The fourth culture, with a twenty-four hour interval, was negative. At the end of the forty-eight hours it was negative; examined again at the end of fifty-six hours it was found positive. The first three cultures, examined at longer intervals, might have been found positive. In such cases there is probably a well-organized clot at the point of infection, with few bacteria thrown off, which may be killed by the bactericidal action of the blood; but later, when the clot begins to disintegrate, a positive blood culture may be obtained. The failure to obtain an early positive blood culture may also be explained on the basis of the virulence of the infection. In the case cited there was no clinical evidence whatever to lead to a diagnosis of infection of the sinus, yet an organized clot was found, extending from within an inch and a half of the knee downward. The patient made a perfect recovery.

Dr. William K. Rogers, of Columbus, Ohio, said that the value of laboratory information in serious complications of middle ear suppuration could not be over-estimated, but it frequently consumed more time than the case allowed, to

obtain it. It often happened that we had not at hand the services of skilled pathologists and so much time would be lost in obtaining them that we often failed to avail ourselves of the information they might give us. Where conditions admitted of delay or when surgical observation was demanded before resorting to operative measures with a well equipped pathological laboratory at hand it was probable that most of us availed ourselves of its findings as a routine practice; otherwise we did the best we could without its aid in reaching conclusions.

It was interesting, therefore, to hear what Dr. Sondern had said to a method for obtaining any of this information in shorter time and with less complicated technic than we had been accustomed to think is necessary.

Dr. Sondern, in closing the discussion, agreed with Dr. McKernon that at times a blood culture is declared negative when it has not been given time enough to show whether a growth will result. Attenuated organisms often do not grow at all. The pneumococcus, for example, often grows slowly, particularly if not virulent, and four or five days may elapse before evidence of it is obtained. The gonococcus will not grow at all unless specific media are used. As blood culture work progresses, doubtless more complex media will be demanded to demonstrate bacteria which will not grow on the ordinarily used media. In some cases of malignant endocarditis there is an organism which will not grow on the media now generally used.

The method of using blood for culture, in which coagulation has been prevented, is comparatively new and very interesting. If a somewhat larger amount than usual is taken, coagulation prevented, and centrifuged in a high power machine, it may be possible to obtain a sufficient sediment of organisms to make direct microscopic examination of value. As there are usually 30, 40 or even 80 colonies in 1c.c. even in mild infections, it is probable that sufficient evidence may be obtained to make an immediate diagnosis by direct examination, leaving the more accurate details to

be learned from the subsequent result of the culture made at the same time. This method is now being tested and communications concerning it will doubtless appear in the near future. The method of using blood in which coagulation has been prevented, for cultures, has the additional advantage, that the surgeon who begins an operation with no intention of having a blood culture made, is in position to obtain the specimen in the absence of the bacteriologist, and by means of the proper admixture, the subsequent correct planting is made possible.

UNUSUAL METASTASIS IN CASE OF SINUS THROMBOSIS.

By JOHN B. RAE, M. D., New York.

In his paper on "The basal principles of Vaccine Therapy," read before the American Congress, in 1910, Adami, of Montreal, makes the statement that in a case of bacteraemia, it is "extraordinarily rare" to find abscesses developing in the brain. It has never before occurred in the practice of the writer to find such a metastasis, and this case is briefly presented to determine what has been the personal experience of the members of the Society in this respect.

M. S., an Austrian boy, of 16 years, was admitted to the Manhattan Eye, Ear and Throat Hospital, on December 5th, 1910, with a history of having had an attack of influenza two weeks previously. After three days of pain, the right drum membrane ruptured spontaneously, with purulent discharge. Pain was relieved, but had recurred at intervals. Temperature on admission, was 104° F., pulse was 124, and respirations 28. There was little mastoid tenderness, and the appearances within the canal were suggestive rather of the middle-ear condition than of any mastoid involvement.

Gas was administered, the drum membrane freely incised. a culture was made from the canal and material taken for a blood count and a blood culture.

The culture from the canal showed a streptococcic infection. A culture from the antrum, made later, also showed a streptococcus. Unfortunately, through a mistake, the culture taken from the metastatic abscess could not be reported upon. The blood count showed 13,100 white cells with 72% polymorphonuclears. Two days later the count was practically unaltered, 14,400 white cells and 70% polynuclears.

The blood culture, taken on admission, was negative. On the day following admission, the mastoid operation was per-

formed, temperature then being 101.2°, pulse 88, respiration 24. The sinus was far forward, was freely exposed, but showed little change beyond a somewhat dark discoloration.

During the next two days the patient was comfortable. Although there was one rise of temperature to 104°, exploration of the sinus was not considered, in view of the negative blood culture. A second blood culture, however, was asked.

Three days after the mastoid operation the sinus and jugular operation was undertaken on the clinical evidence. The sinus was fully exposed, but was not opened until after resection of the vein. In the neck region there was considerable adenitis and periadenitis, and the vein itself was collapsed and empty above the facial. The sinus contained a disintegrating clot. Free hemorrhage was obtained from the torcular end.

Patient had a chill after being returned to bed, but passed a comfortable night. On the day following some redness and swelling developed on the right wrist and a superficial abscess was opened and drained.

The report of the second blood culture now showed a positive streptococcus. The highest temperature on the day after the sinus operation, was 103.4° and on the second day, 102°; on the third and fourth days, 103°, and on each subsequent day, 104°. The remissions varied between 99° and 101°. Eye grounds were normal.

During the night of the third day it was noticed that the patient had some twitching of the right arm and leg, but the clinical note reads that he had a fairly comfortable night. During the following day there was some twitching of the right arm and leg at intervals, but the writer was not fortunate enough to be present during an attack. Patient slept greater part of the night, but had severe twitching again at eight on the following morning. The right side of the face was now also involved.

It is to be noted that all the operative procedures were about the right ear, and that the twitchings which were reported were confined exclusively to the right side. It was,

therefore, very evident that the seat of irritation was the motor area on the left side of the brain, and the question of metastasis there had to be considered.

The patient was seen by the consulting neurologist of the hospital. After examination he reported that operative interference was inadvisable because of the possibility of a coincident vascular lesion and asked for a further opportunity to see the patient. Twenty-four hours later he made a diagnosis of metastasis with meningitis.

Lumbar puncture was immediately made and 30 c. c. of perfectly clear fluid,—but under pressure,—were withdrawn. After centrifugalisation, the sediment showed a few pus cells.

On consultation with Doctor McKernon, it was determined to explore the left motor area. The Fissure of Rolando was located externally, a large flap of scalp elevated, and an attempt was made to raise an osteo-plastic flap. It was found, however, that the diploic veins were enormously enlarged and engorged, and it was deemed wiser to remove the bone with the forceps, as this could be done more rapidly, and the bleeding could be much better controlled. The exposed dura was distinctly hyperaemic. On opening it there was an immediate discharge of pus amounting to, at least, one dram. This pus was not merely subdural but was escaping from a distinct cavity involving the cerebral tissue. The abscess cavity was drained, a dressing applied, and the patient returned to bed. He died during the night. No post-mortem examination was permitted.

While it is possible, in this case, that extension was direct by way of the meninges, it is the opinion of the writer that we had to deal with a genuine metastasis.

A CASE OF SINUS THROMBOSIS.

By FRANK J. BLODGETT, M. D., New York, N. Y.

I am indebted to Dr. Bacon for the privilege of reporting this case of septic sinus thrombosis which occurred in his service at the New York Eye and Ear Infirmary and it is brought to your consideration because it allows an expression of opinion in regard to certain conditions and their treatment.

May 3rd, 1910, G. B., age 34, male, presented himself at the Clinic with the history that two weeks ago he had pain in right ear which kept him awake the first few nights but soon the ear discharged and the pain disappeared. He now comes because of the continued aural discharge. Examination of the ear today, shows a purulent discharge in canal, a drum membrane that is red and swollen with a small perforation in the lower anterior quadrant and slight mastoid tenderness at the tip and no signs of furuncle.

The patient enters the hospital, temperature 98.2 P. 64, R. 16, heart, lungs, kidney, negative, opening in the drum membrane is enlarged, irrigation ordered and he is marked for observation. Examination of smear from middle ear discharge shows streptoccus infection.

May 13th: For last seven days temperature ranges from 98.2 to 99 and there has been a free purulent discharge from canal but today temperature 101, drum membrane condition has not improved and now well marked bulging of the drum and sagging of the posterior superior canal wall is present and marked mastoid tenderness both at the tip and over antrum. A simple mastoid operation is performed, mastoid cells found full of pus and granulation tissue and inner table is perforated, so that sinus wall is exposed, but as the sinus itself appears healthy, nothing more is done than to remove all necrotic bone around the sinus, clean the

wound and dress it with idoform gauze. Examination of smear of pus from mastoid antrum shows streptoccus infection.

May 20th: While being dressed today in the ward by the house surgeon, Dr. Craig, there is found a necrosis of sinus wall. Patient coughed and fainted while being dressed. No pus in lumen of sinus and no hemorrhage from the sinus. Examination of the heart is negative. Examination of the lungs shows friction sound at base right lung, but no dullness and no change in respitory sound, but he complains of pain located here. Blood culture made today is negative but temperature is fluctuating between 99 and 102. No chill or vomiting, but looks septic.

May 22nd: Examination of the lung still gives friction sound at base of right lung, but no area dullness and no roughness respitory murmur.

The temperature range is wider and although the lung is under suspicion, yet the diagnosis of septic sinus thrombosis is made in spite of the negative blood culture and no longer does one consider the possibility of the clot in the sinus being a non-infecting thrombus.

Exsection and ligation of the vein and its branches before any attempt at removal of the septic clot in the sinus is performed and after the vein has been removed, the sinus wall is further exposed and all necrotic areas cut away, a large clot removed from the bulb of the sinus, hemorrhage re-established from each end, sinus packed with idoform gauze, neck dressed open. Cultivation from clot shows streptoccus infection.

From now on, for nearly two weeks there is a septic temperature with wide ranges and the possibility of septic pneumonia or a clot in the inf. pet. sinus or septic endocarditis or lepto meningitis is considered.

May 27th: Today, five days after the exsection of the vein, temp. is 106.2 p. 128 R. 30. Blood culture for the first time is positive. He complains of pain in the left wrist and right leg. There is a distinct swelling and marked tenderness of left wrist. There is a distinct systolic murmur over

the mitral and trispucid area of precordium which is not transmitted. The lungs are still negative. The breath has a very disagreeable odor. He has marked septic appearance, pulse rapid and soft, wound looks well.

May 28th: Hiss Lecocyte Extrace 10 c. c. is ordered to be used subcutaneously every day and all the light nourishment he needs is given him.

June 7th: The right wrist is now the affected joint and the temperature rises to 104 and several small joints in the fingers are swollen and tender. He had a profuse sweat last evening and soda salicyate is ordered with the idea of possible septic endocarditis, but this is the last rise in temperature and the cardiac murmur disappears, all swelling of wrist and fingers subside and he is discharged cured June 28th.

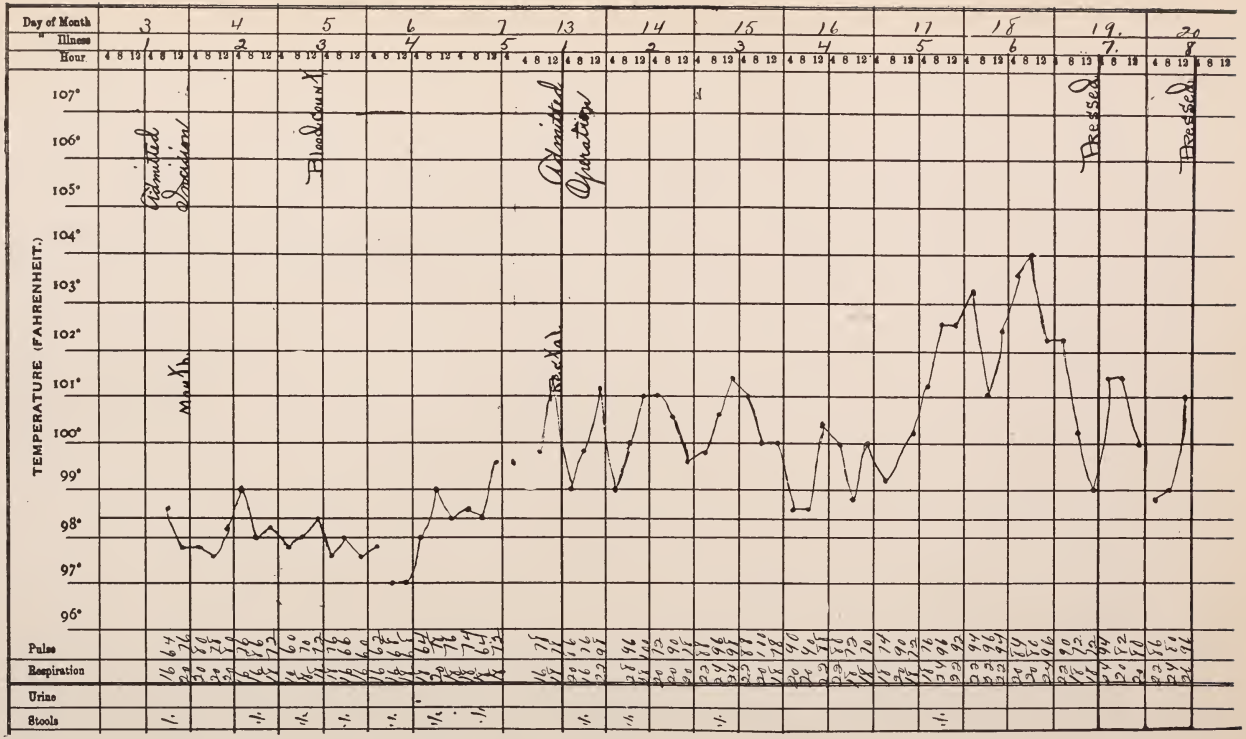
Thrombosis, or the coagulation of the constituents of the blood occurs in both veins and arteries, and is due to the destruction of the white blood cells, which allows the fibrine ferment to be set free, and this allows the union of the calcium and fibrinogen to form fibrine.

The Endothelial coat of artery and vein acts to keep the blood fluid by preventing corpusculor disintegration so that if this coat is not damaged by the toxines, there is no corpusculor disintegration, no fibrine set free, no coagulation of the blood—in a word—no thrombus is formed.

All thrombi are aseptic or septic and once formed, soon undergo changes for the aseptic thrombus is absorbed and replaced by fibrinious connective tissue and the septic thrombus undergoes purulent liquefaction and infecting emboli are set free.

In Sinus Thrombosis the septic germ causes a septic infection of the Endothelial coat of the walls of the veins and the infected walls cause the septic thrombus.

If this is the correct pathology of the formation of a thrombus, then in the case of sinus thrombosis that follows a suppurating ear, i. e. in the presence of germs that have caused a septic process in the middle ear and mastoid cells that has involved the sigmoid sinus.

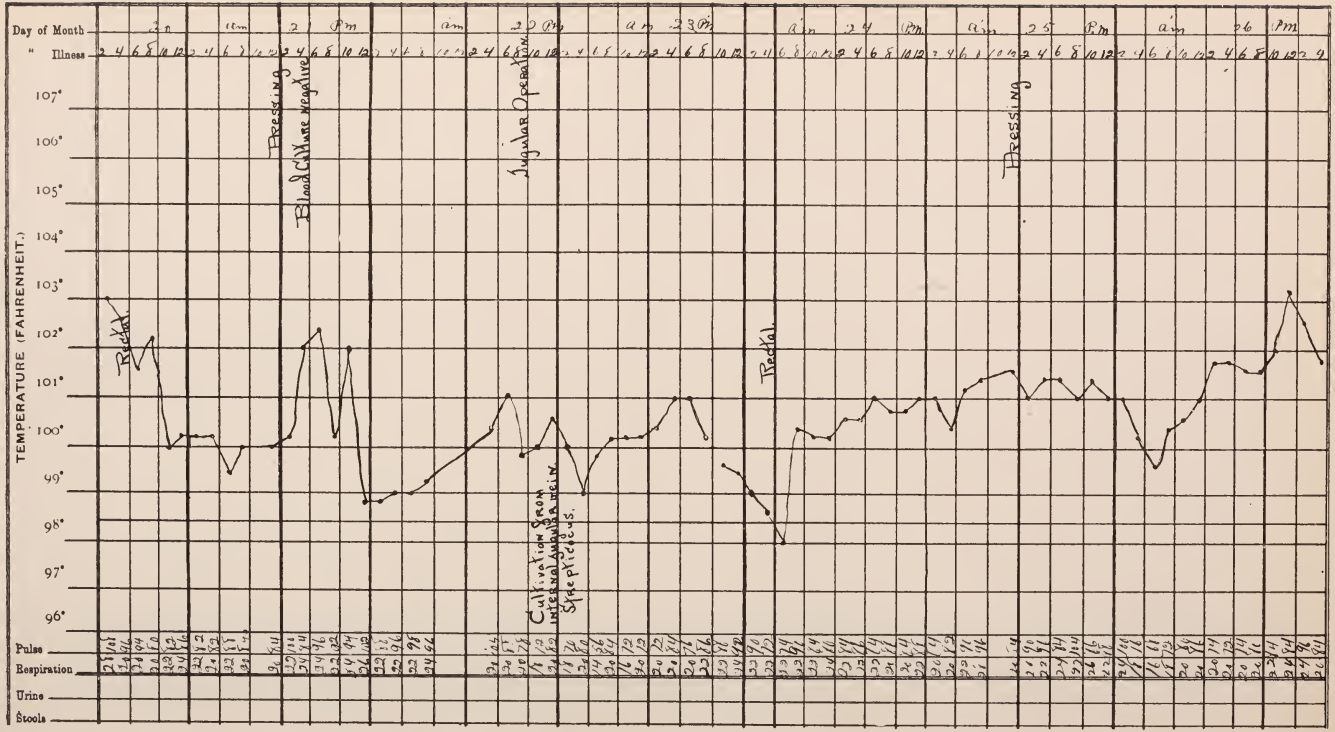


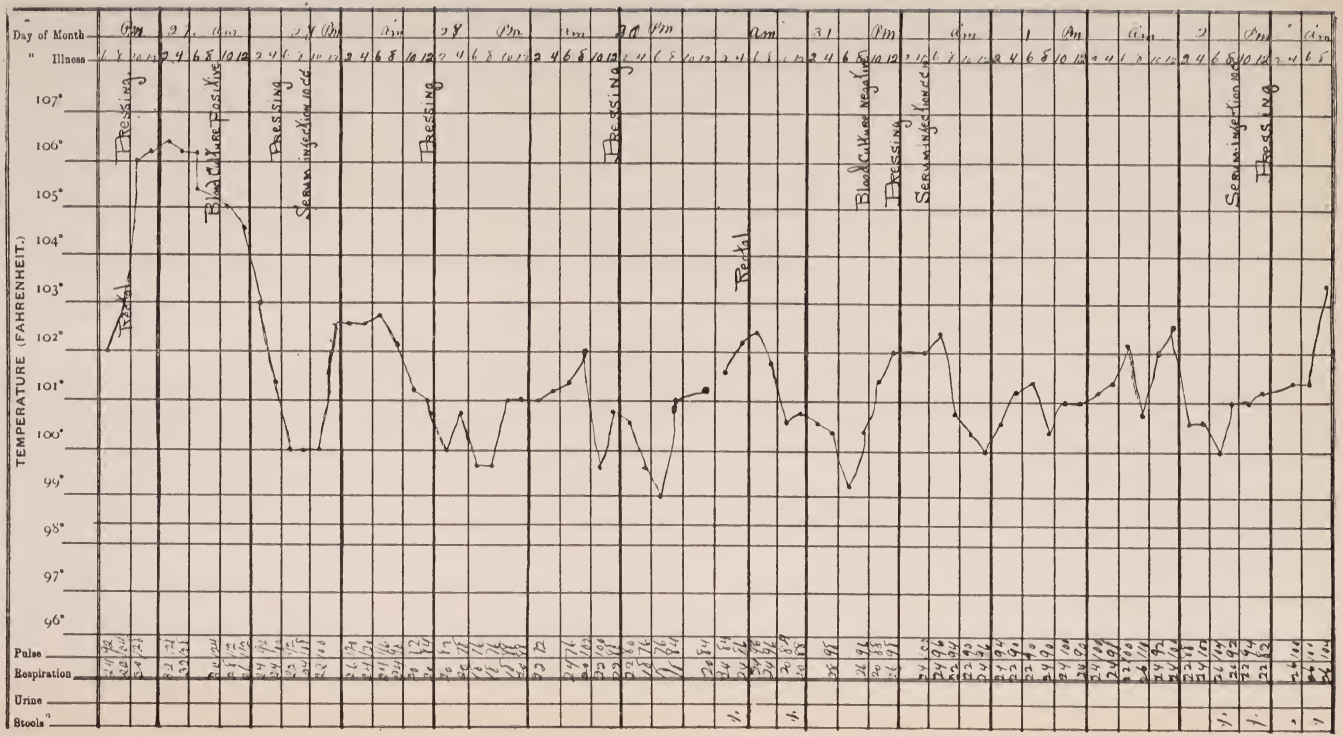
Day of Month
Illness
Hour
TEMPERATURE (FAHRENHEIT.)
107°
106°
105°
104°
103°
102°
101°
100°
99°
98°
97°
96°
Admitted Incision
Mouth
Blood Count
Admitted Operation
Rectal
Pressed
Pressed
Pulse
Respiration
Urine
Stools

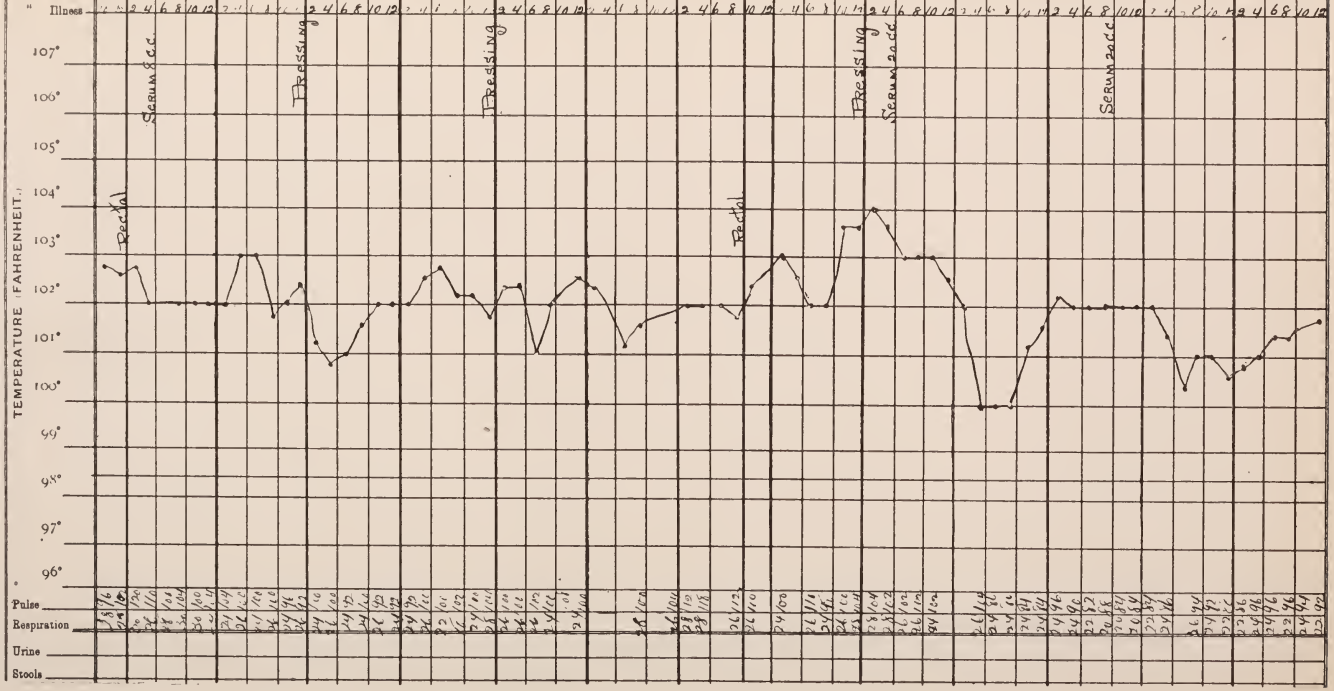
BLOOD CULTURE, MAY 20

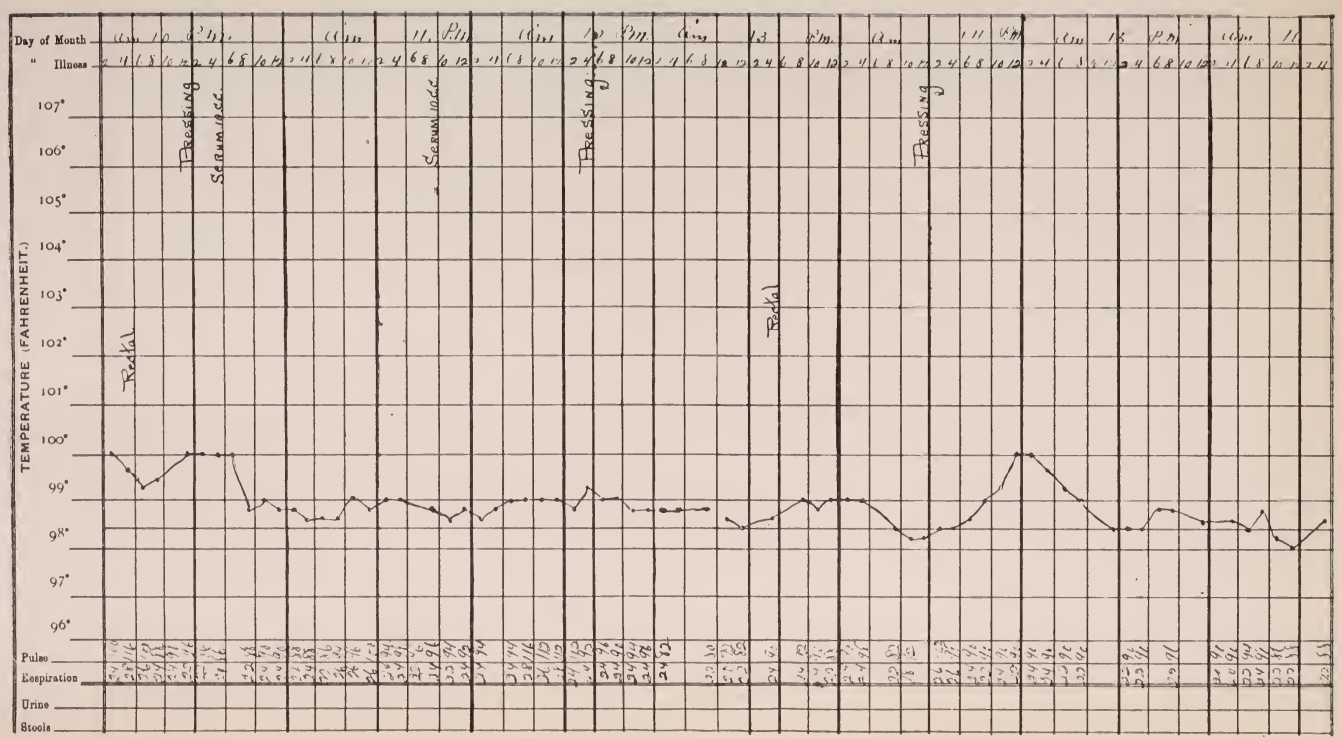
1910.

PATIENT	AGE	SURGEON		
George Biles		Dr Bacon		
ITEM	NORMAL LIMITS	5/21 pm	5/27 pm	6/6 am
RED BLOOD CORPUSCLES	5-6.000.000	5500000	4400000	3040000
WHITE BLOOD CORPUSCLES	5-10.000	10.800	20600	21400
HÆMOGLOBIN	90-100%	— %	— %	— %
MALARIAL PARASITE		none	none	none
DIFFERENTIAL COUNT:				
LEUCOCYTES: SMALL MONONUCLEAR	22. -35. %	18.2 %	10.6 %	15.4 %
LEUCOCYTES: LARGE MONONUCLEAR	2. - 8. %	11.8	2.6	4.8
LEUCOCYTES: TRANSITIONAL	1. - 2. %	2.6	2	2.6
LEUCOCYTES: POLYNUCLEAR	59. -68. %	67.2	86.6	77.0
LEUCOCYTES: EOSINOPHILE	0.2- 4. %	2	0	.2
LEUCOCYTES: MAST CELLS	0. - 0.5%	0	0	0
LEUCOCYTES: MYELOCYTES	0. - 0.	0	0	0
TOTALS		100	98.0	100

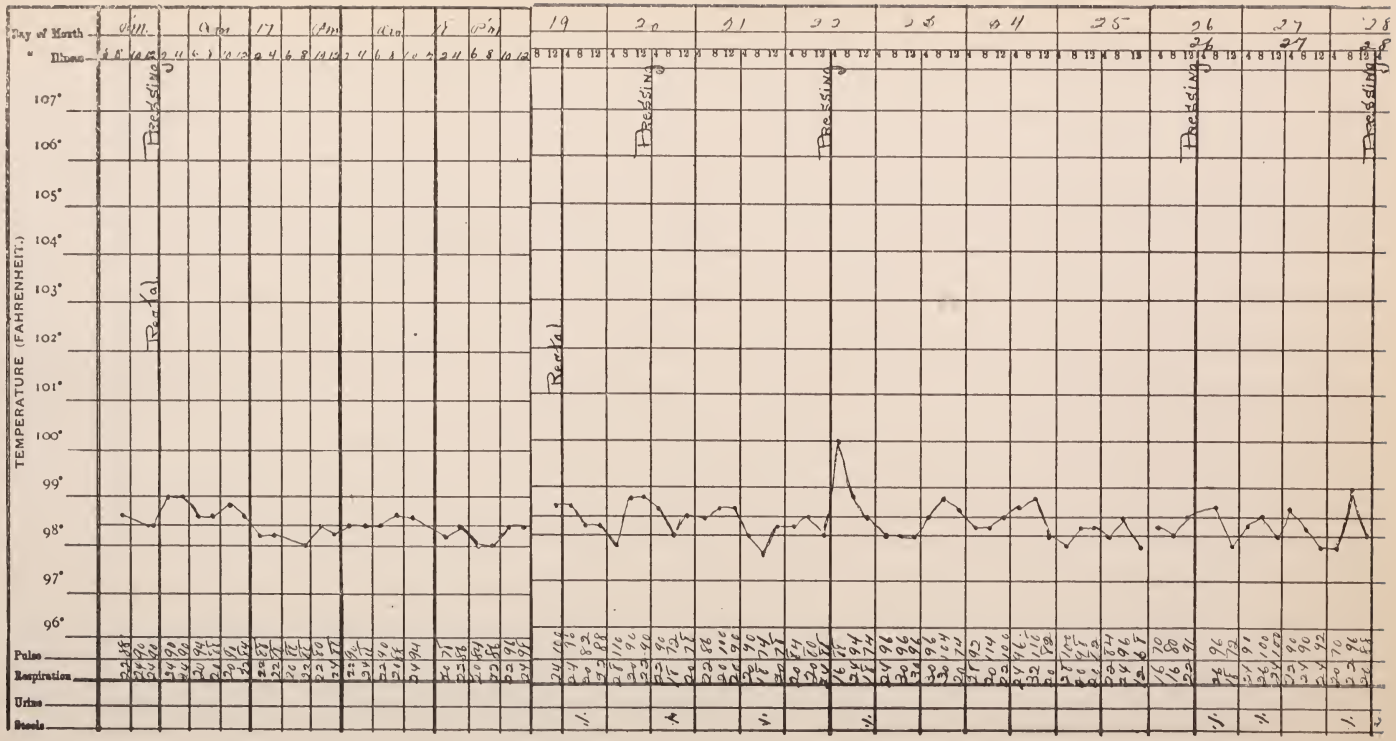

Day of Month
Illness
TEMPERATURE (FAHRENHEIT.)
107°
106°
105°
104°
103°
102°
101°
100°
99°
98°
97°
96°
Rectal
Pressing
Blood Culture negative
Jugular Operation
Cultivation from Internal Jugular vein
Staphylococcus.
Rectal.
Pressing
Pulse
Respiration
Urine
Stools


Day of Month
Illness
TEMPERATURE (FAHRENHEIT)
107°
106°
105°
104°
103°
102°
101°
100°
99°
98°
97°
96°
Pulse
Respiration
Urine
Stools
Dressing
Rectal
Blood Culture Positive
Serum injection
Blood Culture Negative
Dressing


Day of Month
Illness
TEMPERATURE (FAHRENHEIT)
107°
106°
105°
104°
103°
102°
101°
100°
99°
98°
97°
96°
Pulse
Respiration
Urine
Stools
Rectal
Serum 8 c.c.
Dressing
Dressing
Rectal
Dressing
Serum 20 c.c.
Serum 20 c.c.



Day of Month
" Illness
TEMPERATURE (FAHRENHEIT.)
107°
106°
105°
104°
103°
102°
101°
100°
99°
98°
97°
96°
Pulse
Respiration
Urine
Stools
Rectal
Pressing
Serum 10 cc.
Serum 10 cc.
Pressing
Rectal
Pressing


Day of Month
Illness
TEMPERATURE (FAHRENHEIT)
107°
106°
105°
104°
103°
102°
101°
100°
99°
98°
97°
96°
Pressing
Rectal
Pulse
Respiration
Urine
Stools

1. Can this thrombus, whether a fibrinious clot or disorganized and broken down into pus ever be nonseptic.

2. If such thrombi are always septic, and their walls always infected, so will undergo a process of suppuration with the danger of septic emboli, then

Why do we curett the sinus; or

3. In case of necrosis of sinus wall, why should we wait for chills and signs of septic temperature before we remove the vein.

4. And after the vein has been removed why should we sew the neck up; are the tissues of the neck not already infected? And

5. What guide have we to decide whether a case of sinus thrombosis will recover by opening the sinus only, or a removal of the vein is necessary.

After excluding all other suppurating foci, the relative value of clinical and constitutional symptoms, versus Bacteraemia in the **diagnosis** of sinus thrombosis is well established, but in many cases, especially in the all important early stage, often the only positive proof of clot in the sinus is to open the sinus.

Primary Removal of the vein (before opening the sinus), is warranted in all cases irrespective of the location of the clot, in stage "2" and "3" of septic thrombosis, where pyaemic temperature and pus liquefaction give rise to the danger of septic emboli metastasis. But, generally, removal of vein is a secondary operation following opening of the sinus and the only evidence in favor of vein itself being involved is tenderness and oedema of the upper part of posterior cervical triangle in the neck with tenderness of the vein showing a purulent phlebitis.

So that what we really must consider as our guide to decide whether the case will recover by opening the sinus only, or needs removal of the vein, is:

First. The condition of the sigmoid grove and sinus wall, both at the time of the original mastoid operation and during the subsequent treatment.

Second. The results found on opening the sinus.

Third. The degree of infection as shown by prominence of symptoms of sinus thrombosis and blood culture.

A. First—As Regards the Sigmoid Grove. (I quote from an article of Dr. McKernon). 1. A dark sigmoid grove that is covered by soft gritty bone or a necrotic sigmoid grove where the sinus is exposed, demands further removal of the bone to allow examination of the sinus wall, for if we find that the sinus wall is easily compressed by the finger and when the pressure is removed, it fails to refill quickly, we feel warranted in opening the sinus to decide the presence or absence of a clot, and (2) during the subsequent dressing of the mastoid wound after the original operation, we should note the amount and character of the secretion that bathes the wound and the color and consistency of the granulation that covers the bone and especially the granulations over the sigmoid grove, for if after a few days of apparently healthy action, we find discharge diminishes, granulation lose their pink color and become pale and flabby, or over the sigmoid grove the granulations seem to have disappeared, and the grove itself appears dark, especially if now the temperature rises and condition of the patient fails to improve, the sinus should be opened.

B. As Regards the Sinus Walls, we see cases of

First. Pulsating sinus wall, i. e. blood still flows. Here the pulsation may be transmitted from the brain or there is a possible parietal clot, but pulsation is not generally an indication of clot in sinus and so is no warrant to open the sinus.

Second. Necrosis and sloughing of sinus wall. If the clot is septic and the walls are infected, are we warranted

in simply cutting away the sloughing wall and then waiting for chill and temperature fluctuation before opening the sinus or removing the vein.

Third. Respiratory collapse of sinus wall.

Here, with every inspiration the sinus wall collapses, and with every expiration the wall dilates, showing the danger of an air embolis forming on opening the sinus and indicating a clot between the torcular end of the sinus and lungs.

We make pressure with the finger over the sinus wall (at some distance from the point on the sinus that aspirates so that there is no chance of the aspirate being transmitted from the brain or vein) in order to see if the sinus collapses between the Torcular and the bulb.

If the sinus collapses here, we assume the clot is at or beyond the bulb, but we do not exsect the vein until the experiment of lowering the head and making pressure over both veins at the clavicle is made, and if this fails to cause the collapsed sinus wall to refill, then primary removal of the vein is warranted.

Second—As regards the results found on opening the sinus, we see

First—Cases where failure to secure return flow of blood from below, showing complete clot at the bulb and probably extending into the vein or a possible clot of inferior petrosal sinus. Here the blood culture and degree of prominence of clinical and constitutional symptoms of sinus thrombosis will decide the question of the removal of the vein.

Second—Cases where success in securing return flow of blood from below, but here the blood may be from the inferior petrosal sinus and not from the sigmoid sinus, or there may be a partial parietal clot where the blood still flows or the blood may show a purulent liquefaction of septic thrombosis and to prevent septic embolic metastasis, the vein is removed.

In concluding, I would ask an expression of opinion as to the possibility in any case of suppurating ear that is complicated by sinus thrombosis of the thrombus ever being non-septic, and if the walls are always infected and cause the septic thrombus and so give rise to the danger of septic emboli, then should not our future operations by determined more by this fact and less by the success or failure to remove the clot.

So that as soon as the diagnosis of sinus thrombosis is made; it is not now a question of the clot being in the sinus only or extending into the vein; nor is it a question of success or failure to remove the septic thrombus in the sinus, but it is a warrant for the primary ligation of the vein.

Discussion.

Dr. B. Alexander Randall, of Philadelphia, thanked Dr. Blodgett for the careful and guarded statement of the matter. He referred in this connection to Dr. Reik's paper of some years ago, and to his own reiterations for a dozen years on this subject. The question might be asked: Is the case an acute or chronic suppuration? If there be an acute phlebo-thrombosis of the lateral sinus it will often go on to spontaneous closure of the channel, freeing the surgeon from the necessity of jugular ligation. He had seen many cases which he was fully convinced were of that nature. He did not claim that jugular thrombosis of an acute character could not go on to fatal issue, but he believed the acute cases to be far less frequently fatal than the chronic. Dr. Crockett's series of cases showed good results from ligation alone, without evisceration of the vessel, with very little done to the sinus walls and the cells. Whether the case is acute or chronic should be the best criterion as to ligation. In chronic cases, whatever the indications, it is safer to ligate, and, if conditions call for it, widely to exsect the jugular.

Dr. Charles W. Richardson, of Washington, D. C., spoke of Dr. Rae's case. It seemed unusual for metastasis to take place in the brain. While he did not wish to question the correctness of Dr. Rae's conclusions, he must confess that, in view of the manifold avenues of infection, the case seemed to him to be a primary or direct rather than a metastatic condition.

The observations concerning ligation of the jugular were well taken, to a certain extent. When there is a bulbar clot exsection seemed to him better than ligation. He had recently had a case, however, which seemed to controvert this statement. The source of further infection is so great that simple ligation below would leave the patient in so much danger that it would be wiser to excise above as well as to ligate. Three weeks ago he had a case of infection in which the patient had had five chills and five rises of temperature to 104° or 105° before he saw him. It was a chronic case, developing on an old military infection. The patient was operated upon on the morning after being first seen by him. He had a blood count of only twelve hundred; he did not recall the differential, and no cultures were made. The indications were very clear for operation,—chills, elevation of temperature, mastoid tenderness, and pain along the course of the jugular. The eye grounds were normal. On opening the mastoid there was an outpouring, under pressure, of a very offensive pus. Clearing the field of the purulent discharge, the vein was seen to be right under the opening made into the antrum, and pushed up well under the anterior wall of the antrum mastoideum, filling it completely. The vein showed all the evidences of clot, and was immediately opened. A well organized clot was found. It was curetted down toward the bulb, and practically no return circulation was obtained. The jugular was excised for about three inches of its length—about up to the entrance of the facial vein. The patient made the most perfect recovery he had ever seen. The neck wound healed completely by primary union except the lower portion, and the drain was replaced only twice. The operation

was nineteen days ago, and the neck wound had entirely closed when he left home. The patient had no more evidences of disturbance after the operation than after an ordinary mastoid operation. The highest temperature was 100 2-10°. This case was cited as an unusual recovery after infective sinus thrombosis. Unfortunately the clot, which was sent to the laboratory for bacteriological examination was mistaken for tissue and was not examined for micro-organisms.

Dr. James F. McCaw, of Watertown, N. Y., cited a case, in line with Dr. Richardson's remarks, seen by him a few years ago. The patient, a young girl, fourteen or fifteen years of age, had been suffering from measles. The attending physician gave a history of purulent discharge from the middle ear, followed by some tenderness over the mastoid five weeks before. This continued from "bad to worse" as the physician expressed it and five or six weeks after this condition appeared, the speaker saw the patient. According to the attending physician, she had been running a more or less even temperature of 102° to 103°, no exaggerated rises had been noted. When the speaker saw the case, there was an enormous tumor over the mastoid, involving the entire area and pitting deeply on pressure; the child had a yellow waxy look. So far as could be determined, there was no evidence of sinus complication. The ordinary mastoid operation was done and much to his surprise, there was found on opening the mastoid not only the mastoid cells and external table, but the internal table and the walls of the lateral sinus were a mass of broken down tissue, forming one continuous abscess cavity extending nearly to the torcular and down to the bulb. He opened up freely, cleaned out the necrosed tissue, with curette, but was careful not to disturb the clot at either end of the sinus. There was no bleeding either from the bulb or the torcular region. The wound was packed as in any mastoid case. Fortunately the child's temperature dropped to 99½° the next morning and nothing further occurred. This case was recited not as giving evi-

dence in favor of this kind of treatment, but as an unusual recovery.

It may have been one of the cases in which the clot was sufficiently formed to resist the inroads of the disease. He had never had to do a more extensive dissection on the mastoid.

Dr. H. A. Alderton, of Brooklyn, N. Y., said the condition which Dr. McCaw reported is frequently encountered after scarlet fever. He had reported one such case in which nature performed the operation. These cases are particularly apt to occur following the exanthemata, after from forty to sixty days. He had seen a number of such cases, and all that was necessary was to evacuate the pus. In the case cited there was no clinical evidence of the condition.

Dr. Blodgett, in closing the discussion, thanked Dr. Randall for calling his attention to the difference between acute and chronic cases.

Dr. Rae, in closing the discussion, agreed with Dr. Richardson that this unusual condition left one in considerable doubt as to whether it was a direct extension or a metastasis. This patient had been observed from day to day, and no evidence of meningitis found. The day before the operation for the metastasis the patient had been seen by a consulting neurologist, who made the diagnosis of possible vascular disturbance, but made no diagnosis of meningitis. The only sign of meningitis the speaker found was the temperature. Dr. Richardson seemed to think the vein was merely ligated in this case: it was not ligated but excised.

PHYSIOLOGY OF THE COCHLEA.

By ERNEST DE WOLFE WALES, M. D., Indianapolis, Ind.

The physiology of the cochlea is rich in problems. If the function of the cochlea is based on physical laws, it is possible to work out its mechanical principles, and reproduce a working model of the cochlea.

The cochlea is purely a receptive organ. It is not a tone producer, in this way resembling the graphophone rather than the phonograph. It receives sounds within certain limits of pitch and intensity. It receives vibratory pressures, and the larynx by command of the will, or by reflex action reproduces them. A person who has never heard sound cannot reproduce it intelligently, and cannot talk or sing except by special training. Reflexly, like the lower animals, he may make sounds expressing fright, pain, pleasure, or surprise. The male and female of each species giving utterance to high and low sounds, at least, hears a range of such sounds, otherwise his utterances would be of no vital or practical use to him. This statement applies to most animals, birds, and reptiles with the exception of fishes. The highest screams made by children, according to McKendrick and Gray is about 6,000 v. s., yet they hear tones as high as 40,000 v. s. Sounds are sometimes made independent of hearing, for example, the vibrations of a bee's wings produce the tone C when the wings make 256 vibrations a second, but this is no reason that the bee hears his own buzz. These sounds are not utterances; the rapid movement of the wings is made to support the body weight in air. In other words, the bee buzzes in flying.

Do fish hear? This question may be solved by answering another question; do fish utter sounds? Does the whale utter sound? If so he must receive sound by way of the cochlea. Do dancing mice utter sounds? I have never

heard them and I would therefore conclude that they are deaf-mutes. In general the law is, that creatures receiving sound, make use of it by intelligent reproduction, and if creatures do not produce sound by the larynx or some special organ, they do not hear sounds.

Whatever the mechanism of the cochlea is, it is reasonable to infer that it must register the physical properties of sound. This is shown by the following experiment. Take two tuning forks of 256 v. s. and change one of the forks so that the pitch will differ slightly. Set both forks in vibration and hold both to the right ear. The beats are clearly heard. This is purely a physical phenomenon, the cochlea registers the beats, the left cochlea does not record the beats. Now set each fork in vibration and hold one fork to the right ear and one to the left. The beats are again heard, clearly, as before, but in the latter case the physical property of each fork is registered by each organ of Corti to be blended or fused by the brain. In the first case a fusion takes place in the world without, while in the latter case the fusion takes place in the brain, provided each cochlea registers 256 v. s.

The physical properties of sound are pitch, determined by the number of vibrations per second; intensity, determined by the amplitude of the wave, the higher the wave the louder the sound theoretically; and lastly the color-tone called timbre, quality, or character of tone, which depends on the form of the wave. These properties produce stimuli which cause sensation at the terminal hair cells on the organ of corti.

Sound is the result of variation in pressure, these differences in pressure cause vibration of an elastic body such as air. When these vibrations are regular we call them tones, but when irregular, noises. The organ of hearing is a receptive organ for the multitude of tones varying in quality, pitch, and intensity, and the varying noises in the immediate world about us. These sounds are heard if of sufficient intensity and within the limits of 40 to 40,000 single vibrations a second. The cochlea does not reproduce

these sounds but the energy that the sound possesses causes pressure stimuli; a feeling, received by the sensory hairs of the organ of corti which produces a sensation, which in turn is carried along the nerve fibres to the brain and there perceived. The cochlea contains an impressible apparatus as the wax roll of the graphophone receives the pressures of sound. This impression is not sound, it is the result of sound, and can be reproduced only by a motor apparatus.

To best transmit sound from the air to a liquid, a middle-ear apparatus is necessary. The middle-ear apparatus is like a pendulum in equilibrium, ready to impart vibratory pressures from the outer world to the perilymph. I have tried to show how this equilibrium is maintained by the intrinsic middle-ear muscles in a physiological manner, in a former paper read before the American Laryngological, Rhinological and Otological Society at Chicago, February, 1909. These vibratory pressures could not be registered if it were not for the fact that the perilymph is capable of mobility. Mobility of the perilymph is equal to the limits of excursion of its vestibular and cochlea membranes. Vibratory pressures are transmitted and not conducted from the membrani tympani to the vestibular membrane by the ossicles. Articulations of the ossicles still further prevent conduction. Transmission implies the possibility of molar movement while conduction implies the possibility of molecular movement. Molar movement of vibratory pressures is limited between 40 and 40,000 v. s. a second. Outside these limits we do not feel vibrations by way of the cochlea. The limited excursion of the vestibular membrane may limit the lower vibratory pressures, while lack of physical delicacy may limit the higher vibratory pressures. If there is no excursion of the vestibular window, as in complete ankylosis of the stapes, there is no mobility of the perilymph, and if there is no mobility of the perilymph an individual is totally deaf. Ankylosis is a relative term in otology. It may be partial or complete. If there is any hearing, there is some mobility. In any hinderance to the normal movement of the stapes the coarsest and finest movements natu-

rally suffer most, hence the lowest and highest tones are not heard. It is well established that occlusion of the niche to the round window and ankylosis of the stapes causes total deafness. Mobility of the cochlea fluid may be possible through the aqueductus cochlea or saccus endolymphaticus, but this seems doubtful. Where there is hindered mobility as in oto-sclerosis, patients claim to hear better when riding in a car or when in a noisy place. This is probably not true, but only apparent, for on a train we naturally raise our voices in talking, and we generally sit close to the deaf person with whom we converse. We must speak loud enough to hear our own voice, or we cannot expect to make our traveling companion hear in a noise, even if he has normal hearing. The voice to be heard must be of greater intensity than the noise unless there is considerable difference in pitch. Such persons who have hindered excursion of the stapes, and hence lessened mobility, hear better when the sound received is intense, whether in a quiet room or in a noisy place.

The whale presents an interesting study. If the whale has complete ankylosis of the stapes, he must be deaf to sound when he comes to the surface of the ocean, while he may have a limited motion of the stapes at great depths allowing some mobility.

It is necessary to admit that the function of the cochlea is hearing. No cochlea, no hearing. Fishes have no cochlea and therefore do not hear. In the development of the cochlea there is at first a budding out from the otocyst, a lagena as seen in reptiles and birds, and later, higher in the animal series this slightly curved portion develops into a true cochlea. Birds and reptiles with limited outgrowth of this special organ probably have a limited sense of hearing, and judging from the high sounds birds and reptiles make, we expect only a development of the beginning of the cochlea whorl. It is reasonable to think that the greater the number of turns of the cochlea the greater the field of hearing.

Granting that mobility of the cochlea fluid is necessary for sound transmission, it is essential to consider some anatom-

ical facts of the cochlea and apply the laws of physics. The cochlea duct is roughly speaking a triangular shaped receiver which tapers like many of the wind instruments; this receiver of sound first receives its energy from the end of smallest dimensions and is similar to the flute. The nearer the source of motive power the higher the pitch, the further removed the lower the pitch. Two sides of this triangular shaped tube are composed of membranes, while the third side is a part of the spiral ligament, which serves for the attachment of the basement membrane. Reissner's membrane, the membraneous side toward the scala vestibuli, is the simplest membrane and is very delicate, capable of transmitting vibratory pressures from the perilymph to the endolymph within the triangular tube. The thicker and more tense a membrane, the less its power of shock transmission from one fluid to another. If this triangular duct had walls of thick or even thin bone lacking flexibility, the fluid within its walls would not receive slight vibratory shocks, and any pressures or impulses started up from movements of the stapes in the oval window, would keep a course along the scala vestibuli, through the heliocotrema to the scala tympani, and cause movements of the round window membrane corresponding to movements of the oval window, and therefore would not transmit shocks to the endolymph. Since Reissner's membrane is not tense or thick but very delicate, vibratory shocks are readily transmitted to the fluid within the cochlea duct, where they cause the tectorial membrane to vibrate and thus stimulate the auditory hairs. The sound impulses do not necessarily pass by way of the heliocotrema but may take a short cut through the basement membrane to the scala tympani. The basement membrane is much thicker and weighted by the organ of Corti. This membrane is attached to the lower border of the osseous spiral lamina, supports the organ of Corti and curves sharply upward at a point beginning with the attachment of the scala tympani portion of the spiral ligament, and ends near the vas prominens. Of what use is this triangular bimembraneous tube within a tube? If the base-

ment membrane is important so is Reissner's membrane, for it forms the bimembraneous mute flute. This mute flute is affected by the tones received, in the same manner as if the tube itself were the producer of tone, and is therefore naturally selective of vibratory pressures, the high pitched tones affecting the end nearest their entrance and the low pitched tones affecting the end farthest from the source. Thus the vibratory pressures received, cause a sympathetic vibration of the fluid within the tube, and this is imparted to the sensory hairs in that region of sympathetic response, producing a sensation. The number of tones or noises received is unlimited within the limits of hearing. Noises may be considered as a definite fixed jumble of tones. For example, if a new machine were invented, it would make a characteristic noise, which we would associate with that machine, just as we know the whirr of the flying machine, the chug, or hum, of the automobile, the noise of the mowing machine and numerous other machines, and thus all noises have a definite physical character or tone color. Between 256 and 512 v. s., over 1,000 different tones may be distinguished according to Ebbinghaus. Possibly in all 385,000 tones may be differentiated or as many sensory hairs as there are found in Corti's organ. Differences of tone are harder to distinguish, the nearer we approach the high or the low limits of hearing. The cochlea duct is therefore a reversal of the flute. The flute or flute organ pipe is a producer of tone, the cochlea duct a receiver of tones. In the same sense a graphophone is a reversal of the phonograph. If sensation of pitch can be accounted for mechanically by a flute theory, intensity is but an increased stimulus, and the combination of pitch and intensity will account for color-tone.

The advantage of such a theory over the piano theory of Helmholtz is, that we can account for an unlimited number of tones within the limits of hearing. We are not confined to a given number of fibres. Then again, the vibration is received within the closed tube containing the auditory hairs which are the natural sensory terminals of the cochlea nerve.

These hairs must be irritated and not the fibres of v. Henson in the basement membrane, which are not directly connected with the nerve endings and thus further present mechanical difficulties. Islands of hearing may be accounted for by a flute theory, for we are considering the vibration of a column of fluid and not the fibres in a basement membrane, weighted and further complicated with blood-vessels and longitudinal fibres.

Still further delving into speculative otology, each anatomical peculiarity of the structure of the cochlea must have a reason for existence. Take, for example the spiral arrangement of the cochlea. It would seem as if all sound must travel with a spiral movement to produce such a winding staircase. The old experiment of the wire spring to represent how sound travels in a to and fro manner without propulsion of the individual molecules of air. If sound does not travel with a spiral movement, how could sound develop the cochlea; and if not sound what other energy could cause the development of the cochlea in a spiral course? The higher the pitch of the sound the closer the spirals come together, and the nearer the sound spiral approaches a solid straight line in any direction. Is this the reason why high tones are more penetrating? The lower the pitch of the sound the further apart are the spirals; the diameter of the spiral would represent the intensity. Sound is therefore a spiral vibratory pressure.

It is a question if we should make fun of the men in Aristotle's time who believed that the high pitched sounds traveled faster than the low pitched sounds. Experiments in the velocity of sound vary greatly in air and liquid due to the variations in heat, air currents and imperfect recording instruments. Let us imagine that the difference between the velocity of the highest audible tone and the lowest audible tone is one foot a second, in other words a tone of 40,000 v. s. traveled 1,101 feet a second and a tone of 40 v. s. traveled 1,100 feet a second, the tones between these extremes traveling in proportional velocities according to their pitch. Now the length of the cochlea duct (one and two-

fifths inches in the human cochlea) with its sensory hairs may have some relation to the difference in velocity of sound from the highest to the lowest. Although we cannot estimate or comprehend time to minute fractions of a second, the series of hair cells may be able to make such physical records. The rods of Corti with the basement membrane forms the tunnel of Corti, which is like the wedge piece of an organ pipe. This wedge shaped column of fluid separates an inner single row of sensory hair cells from three, four, or five rows of outer sensory hair cells. The purpose of this arrangement is not yet accounted for in the peripheral analysis of sound. The arrangement of hairs in the sensory hair cells in straight rows and horse-shoe arrangement is probably not arbitrary but of a physical necessity. The tubules of Shambaugh may be emptied of fluid by compression when the blood vessels of the stria vascularis and spiral ligament are filled with blood, when we are active, but in sleep, when anæmia of the brain takes place, these tubules may take in fluid and so lessen the amount of endolymph in the cochlea duct, allowing the perilymph to press Reissner's membrane on the tectorial membrane and thus act as a dampner to incoming sounds, just as the eyelids help to keep out light when we go to sleep. Thus the tectorial membrane may be a factor in the complex problem of sleep by shutting out tone sensation. Lessened amount of blood in the spiral ligament may be sufficient to lessen the tension on the basement membrane causing a collapse of the ductus cochlearis.

Whatever theory is accepted must conform with the anatomical structure, as well as the laws of physics. The Helmholtz theory does conform with most of the physical laws of sound so far as we know them. It conforms with one of the anatomical facts, namely the variation in the length of the fibres of v. Hensen in the basement membrane. Helmholtz has the key to the physical requirements but it does not seem to fit the anatomical findings. The Helmholtz theory does not require a tube within a tube, hence the endolymph would seem superfluous. The function of the

tectorial membrane, the necessity of the rods of Corti in the higher mammals, the tunnel of Corti, and the inner and outer hair cells; all these anatomical structures with the exception of the spiral ligament to vary the tension of the fibres of v. Hensen seem unnecessary for the peripheral analysis of sound by the piano theory of Helmholtz. Some theory which would give every structure of this wonderful analyzer of sound, an action, would be nearer the truth. The cochlea has been moulded by the physical effects of sound through centuries of development, hence every curve and every structure has its purpose. This suggests that variations in structure may be hereditary, and play a part in otosclerosis. Faulty structure would cause the spiral vibratory pressures (sound) to deviate from the proper channels and bring about mechanical iritation causing the early stage of hyperaemia to be followed by the osteoporosis. This problem and others are awaiting solution through animal experimentation. If there was no sound, there would be no cochlea, and if no cochlea there would be no hearing.

Discussion.

Dr. B. Alexander Randall, of Philadelphia, did not agree with the reader of the paper on certain points. He asked which cochlea was meant when the hearing of the "beats in the cochlea" was mentioned. He considered it central, not cochlear.

With reference to the question of paracusis Willisii he said that deep tones are to normal ears those which have the greatest force and tend to over-ride and blur out the higher voice-tones, especially the subtle consonant sounds; but they are practically unheard by catarrhally deaf individuals, and therefore do not interfere with their hearing. The normal listener close at hand may be unable to understand what is spoken in his ear on the train, while the deaf person will hear it six seats away. The improved hearing in paracusis may be described as profitable deafness to low tones.

In nerve-deafness there may be perfect hearing for low tones and limited impairment for high tones, and yet hearing for speech be very poor. Reversing what obtains in catarrhal deafness, normal hearing of low sounds in noisy places overcomes the essential speech inflections.

There were many points of great suggestiveness in the paper, from which he hoped to get a good deal of instruction. It had already brought to his mind many things which were very ill-defined before. The suggestion of the increase in the turns of the cochlea corresponding to the range might be tested with the guinea pig, whose cochlea has very many turns.

There is a good deal of the physiology which the speaker did not expect to discuss, for the reason that in the physics of sound there is yet much which is unknown.

Dr. Wales, in closing the discussion, thanked Dr. Randall for his criticism and agreed with him that the fusion was central, but that the peripheral analysis was different in the two experiments. In the one case with both forks, at one ear there was a peripheral analysis of beats produced without the ear, while in the other case there is a peripheral analysis of single tones producing the same effect of beats by central fusion.

OBSERVATIONS ON INTRA-LABYRINTHINE PRESSURE.

By D. HAROLD WALKER, M. D., Boston, Mass.

Mr. President and Gentlemen:—I regret to report that the experiments which I had hoped to carry out are unfinished, due to the anatomical difficulties encountered in the animals used. The influence of varying circulatory pressures on the intra-labyrinthine pressure, I had hoped to show, also the influence of lumbar puncture on the intra-labyrinthine pressure. If anyone should be interested in these problems, I think the dog, sheep or calf are the animals best suited for the purpose.

SOME EARLY BOOKS ON THE EDUCATION OF THE DEAF.

By FRANCIS R. PACKARD, M. D., of Philadelphia.

There are, in ancient history, several accounts of deaf mutes who were educated so that they could communicate with other persons and understand them.

Jerome Cardan is generally regarded as the first author who stimulated interest in the study of deafness by his writings, but the first man who appears as a practical teacher of the deaf was Pedro de Ponce, or Ponce de Leon, a Spanish monk of Sagahun, who instructed with wonderful results quite a number of pupils. He was born in 1520 and died in 1584.

Juan Pablo Bonet wrote the first treatise on the art of teaching the dumb to speak. It was published at Madrid in 1620, three years prior to his death. It is said that he used signs and a manual alphabet in the beginning, and then depended mainly on writing and articulation. It is probable that Bonet was the priest whom Sir Kenelm Digby refers to as the instructor of the Spanish deaf lord that was exhibited to him in Spain.

Holger Mygind (1. Deaf Mutism, 1904) states that according to another Spanish monk, Benito Geronymo Feyjoo, the material from which Bonet wrote his book was stolen from one of Ponce's pupils. He calls him a plagiarist and an imposter.

A contemporary and countryman of Bonet was Ramirez de Carion, who was also a successful instructor of the deaf.

Dr. Seiss (Children of Silence, Phila. 1887) states that in France there was a deafmute school conducted at Amiens in 1746, by one Etienne de Fay, who was himself deaf from birth. A Portuguese, Roderiguez Pereire, taught the deaf

in France and in 1749 was given a hearing before the Royal Academy of Sciences in Paris, which reported favorably of his work. In 1760, Abbé De l'Epee began his work in the education of the deaf and dumb. He established a seminary for their gratuitous education, in which he was aided by the Abbé Sicard who carried on his work after De l'Epee's death. De l'Epee and Sicard used a manual method, although they also used as an auxiliary the oral method.

The first book which I wish to show is, so far as I am aware, the earliest publication in English dealing with an oral method of instruction for deaf mutes. It is entitled—

"Philocophus: or the Deafe and Dumbe Mans Friend. Exhibiting the Philosophical verity of that subtile Art, which may inable one with an observant Eie, to Heare what any man speaks by the moving of his lips. Upon the same ground, with the advantage of an Historicall Exemplification, apparently proving, That a Man borne Deafe and Dumbe, may be taught to Heare the sound of Words with his Eie, & thence learn to speake with his Tongue. By I. B. surnamed Chirosopher.

Sic canimus Surdis————

London, Printed for Humphrey Moseley, and are to be sold at his shop in Pauls Churchyard, 1648."

Bulwer had previously published a book entitled, "Cheirologia; or the Naturall Language of the Hand," from which circumstance he derived the surname "Chirosopher."

The Deaf and Dumbmans friend is made up of several parts. Following the dedications and introductions we find—

"A Table of such hints and notions as more directly concern Deafe and Dumbe men." These have the appearance of being notes or jottings which the author used or intended to use in elaborating lectures or instructions for the pupils in the Academy which he thought should be established for the instruction of the deaf and dumb. Many of these "hints and notions" have a very modern sound, thus—

"Why Deafe men beget Deafe children"—"Why the children of Deafe men are not always Deafe."

"The causes why many in a Family prove often times Deafe and Dumbe, very darke and obscure."

"Why those who are born Deafe are consequently Dumbe."

"The chief signe to distinguish naturall Deafeness, from that which happens through a disease."

"That Dumbe folks, when they are angry, will make a very great gabling noyse."

"The sad and lamentable condition of those, who are borne Deafe and Dumbe."

"The sadder condition of those who are Deafe and Blinde."

"The yet more miserable condition of those who are Deafe, Dumbe, and Blind."

The second part of the treatise is a very learned and elaborate discussion of language and utterance. The third part begins by quoting the description by Sir Kenelm Digby of a Spanish lord whom he had seen while in Spain with Prince Charles, who although a deaf mute had been taught to understand and express himself so well that he could interpret by reading the lips the sounds of a foreign tongue, and render them back with great accuracy. The conclusion of Bulwer's work is devoted to a minute analysis of Digby's narrative in which he places the most implicit confidence, explaining at length the reasons why such a phenomenon could be brought about.

The second book which I show is, "Alphabeti verè Naturalist Hebraici brevissinis Delineatis. Qua simul Methodum suppedirat, juxta quam qui surdi nati sunt sic informari possunt, ut non alias saltem loquentes intelligant, sed & ipsi ad sermonis usum perveniant. In lucem edita à F. M. B. ab Helmont. Sulzbaci, Typhis Abrahami Lichtenthaleri. A. D. 1657."

The subtitle being translated reads, "Illustrating the method by which deaf mutes may not only understand each other, but may also themselves speak." This little work in

Latin contains an exposition of an oral method, which in a primitive way is based in many particulars on the principles and facts used at the present time. The frontispiece represents the author studying and delineating the position of the lips in utterance. At the end of the book are some thirty odd illustrations representing the various positions assumed by the lips and tongue in articulation; also some pictures of physical apparatus employed in the author's researches.

This copy of Helmont's book is from the library of the Abbe Fauvel. Helmont was a Dutchman, and in 1692 John Conrad Amman, a Swiss physician, practicing in Amsterdam, educated a young deaf girl of Haarlem so that she could speak and understand conversation. According to Seiss, (Children of Silence) in 1692, he published an account of his method entitled, "Surdus Loquens," which was republished in an enlarged form, in 1700, under the title, "Dissertatio de Loquela," and was later published in English.

The third book bears the title, "Didascalocophus; or the Deaf and Dumb man's Tutor—to which is added A Discourse of the Nature and Number of Double Consonants: Both which Tracts being the first (for what the author knows) that have been published upon either of the Subjects. By G. Dalgarno—Printed at the Theatre in Oxford—A. D. 1680."

A picture of the Sheldonian Theatre, Oxford, illustrates the title page.

The author was a grammarian and his book is full of the distinctions and rules which rendered so complex all the grammars of his day. He deals with the manual method of speech, explaining his method of teaching it and suggesting its defects, especially the fact that those with whom the deaf man wishes to converse must also know chirology, as the author terms it. He then proceeds to consider the feasibility of an oral method of conversation for the deaf, which in many respects presents a crude prevision of the methods employed in the oral instruction of the deaf today. Dal-

garno certainly grasped many essential facts. Thus he states, "That a Deaf man may be taught to speak, is no more a doubt to me, than that a Blind man may be taught to write"—Again in his Chapter IV, "Of a Deafman's Capacity to understand the speech of others," he says, "That words might be gathered, and read from the transient motions, and configurations, of the mouth (if all the several distinctions of letters, were no less manifest and apparent to the Eye, than to the Ear from the speaker's face) as readily as from permanent characters upon paper, is not to be doubted." He proceeds, however, to reject the oral method because, he states—

"But that all the distinctions that are perceived by the Ear in speaking, cannot especially be perceived by the Eye; I will prove by an argument, which tho it be a Posteriori, yet I hope it will be of evidence and force sufficient, to effect what is thereby intended.

"If the same distinctions of letters and words did appear to the Eye from the motion of the speaker's mouth, which are discernable to the Ear from the articulation of his voice; then it would follow; that the capacity of a Deaf man, would be equal to that of a Dumb (but not Deaf) for learning a language, so far at least as to understand it.

"But the capacity of a Deaf man is not equal to that of a Dumb, for hearing a language from speaking.

"Therefore all the distinction of letters, are not manifest to the Deaf man from the speaker's mouth."

He quotes Sir Kenelm Digby's story of the "Spanish Deaf Lord," who was "able not only to know strange letters in strange languages, instantly, but also to be able to imitate them, tho he had never been taught," and states his disbelief in it. The author then goes on to declare his belief that the "Dilligent Hand" of the tutor is the greatest engine in bringing relief to the deaf.

Dalgarno makes no reference to any previous work on the subject.

Another early English book on the instruction of the deaf is—

"The Deaf and Dumb Man's Discourse or a Treatise Concerning those who are Born Deaf and Dumb, containing a Discovery of their Knowledge or Understanding; as also the method they use, to manifest the sentiments of their mind. Together with An Additional Tract of the Reason and Speech of Inanimate Creatures. By George Sibscota, London, Printed by H. Bruges, for William Crook at the green Dragon without Temple Bar, 1670."

This little book is in the nature of a philosophical discourse and does not treat the subject in any of its practical aspects. The copy which I hold has many notes in manuscript concerning ear trumpets.

Lastly, I have a copy of the famous book of l'Abbè Charles Michel de l'Epee entitled—

"La Veritable Maniere d' instruire Les Sourds et Muets, confirmee par une longue experience—Par M. l'Abbe———, Instituteur des Sourds Muets de Paris. Paris 1784."

These books are all shown from the Library of the College of Physicians of Philadelphia by which they were recently acquired.

A CASE OF RAREFYING OSTEITIS OF THE MASTOID BONE.

By CHARLES W. RICHARDSON, M. D.

(Read before the American Otological Society, June, 1911.)

I present this case of rarefying osteitis of the mastoid bone partly on account of its rarity and partly on account of the fact that this is a unique case in otological practice and on account of the rarity of this type of bone inflammation as affecting the flat bones.

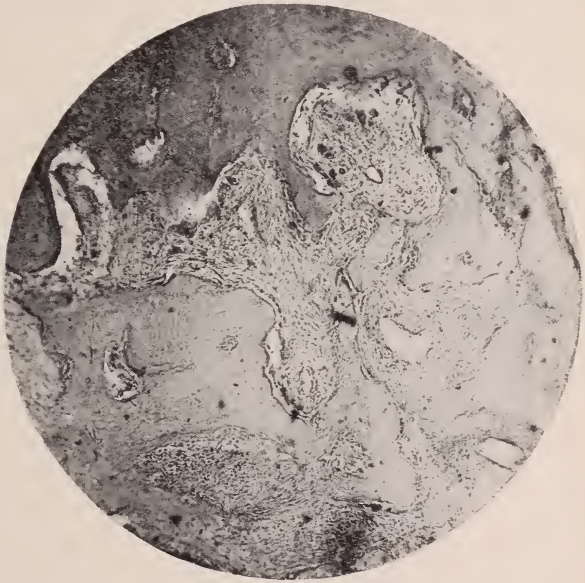
On October 21, 1910, a man of thirty-two years came under my care with the following history: In January, 1910, he developed an acute inflammation in the right middle ear, which subsided after running the usual course. There was a moderate degree of mastoid tenderness, which did not abate with the cessation of the middle ear inflammation. This mastoid tenderness, which was intermittent at first, towards the end of February became continuous, with exacerbations of intense suffering at night. In March he developed what was stated to be an abscess in the canal, which was incised on two separate occasions. There was no relief afforded the patient to the mastoid suffering by the opening of the abscesses in the canal wall; the mastoid pain became more intense with each succeeding month, until the patient was nearly exhausted from pain and loss of sleep, when he came under my observation. The patient states that, while the pain was most intense in the mastoid region, it radiated from this region all over the head. He also stated that large knots formed on his head at night, which would subside during the day. I must confess that, after listening to the patient's history and his description of his intense suffering, I felt quite skeptical as to its truthfulness. During the greater portion of this period he had been under

the observation of several aurists. On examination of the patient I found that I had erred in my opinion. There was a slight infiltration of the soft tissue over the mastoid. The auditory canal and the membrana tympani were normal, the whole mastoid region was exquisitely tender—more so than I had ever observed in any patient before—and the slightest touch with the finger would cause him to cry with pain. This tenderness was limited entirely to the mastoid. The patient would permit considerable pressure beyond the confines of the mastoid without any evidence of suffering. A blood count was made the same day, showing 9,300 white cells with a practically normal differential. The patient's temperature taken at this time was 99°; when taken at the hospital the following morning, it was found to be normal. I was uncertain as to the nature of the condition which here presented itself, but, feeling that relief was demanded, I decided upon doing an exploratory operation the next day. The usual operation procedure was adopted as in doing the ordinary mastoid. After incising the infiltrated soft tissues and raising the periosteum, during which a few drops of pus were seen, the exposed bone over the mastoid presented two peculiar phases, viz., it was of a pinkish color and imparted a decidedly rough sensation to the examining finger. This area of discolored bone also seemed to be slightly raised above the surface of the adjoining healthy bone. The area of diseased bone was chiseled away in every direction and as far inward as the inner table of the bone. It was not deemed necessary to open into the antrum mastodeum. Various chippings and large pieces of bone were saved for microscopic examination. The patient never had a particle of pain after coming out of the anesthetic and made an uninterrupted and rapid recovery.

The pathologist of Providence hospital, Dr. R. A. Hamilton, made the following report on the specimens of bone placed in his hands:

Macroscopic examination of specimen. The bone is redder and much softer than normal, some of the pieces being easily fractured by pressure of the fingers.

Microscopic examination (after decalcification) shows increased production of connective tissue and round cell infiltration, the character and morphology of the cells excluding malignancy. In some of the sections there are areas



where the bone is undergoing absorption, and in others, there seems to be a direct formation of new bone. The microscopic picture is one commonly met with in rarefying osteitis.''

Discussion.

Dr. Norval H. Pierce, of Chicago, asked if bacteriological examination was made, to which Dr. Richardson replied in the negative. Dr. Pierce then said there is a slight similarity to this process in spongification, which is the forerunner of oto-sclerosis. There is a similarity between this and the process which takes place in the ribs of animals, and it would have been interesting to carry on bacteriological examination. He had never seen a case in which this occurred in

the mastoid. He knew, however, that it does occur. He had had patients who complained of extreme tenderness over the mastoid, and in two of these he operated, finding comparatively normal mastoids. After operation the pain entirely ceased. In another case he refused to operate, and after a time, with some treatment directed toward the general health, the pain ceased.

Dr. Ernest de Wolf Wales, of Indianapolis, Ind., asked if there was any history of syphilis in this case.

Dr. Richardson, in closing the discussion, said there was no history of syphilis in this case. A culture was desired and a specimen sent to the laboratory, but for some reason the culture was not made.

THE VERTIGO OF VESTIBULAR PARALYSIS.

By PHILIP D. KERRISON, M. D., New York.

The purpose of this paper is to describe a clinical phase of suppurative labyrinthitis which, so far as the writer knows, has escaped formal identification and description in the literature of the subject, viz.,—the vertigo of the latent, or quiescent, stage of the disease.

Wherever labyrinthine vertigo is mentioned, we find reference to the so-called "vestibular vertigo," or "vertigo of vestibular irritation," so graphically described by Barany. The essential features of this phenomenon,—i. e., its invariable presence at the onset and beginning of the acute stage, its rotary character, i. e., the patient's subjective sense of the rotation of surrounding objects in the same plane as that of the co-incident nystagmus,—these characteristic features are familiar to all of us. As further identifying it, Barany has formulated the following law:— Vertigo of vestibular origin is always accompanied by vestibular nystagmus, and is always increased when the eyes are turned in the direction of the quick nystagmic movement. I personally believe that this contention of Barany is well founded, my reason for this belief being (1) that I have seen no instance of indisputable vestibular vertigo in which vestibular nystagmus was absent; and (2) in studying the phenomena of experimental irritation of the vestibule,—e. g., in the caloric and rotation experiments,—the vertigo, or at least its resulting ataxia, has invariably subsided a little before the nystagmus. But if we accept Barany's law, we must be prepared also to believe that when in suppurative labyrinthitis the spontaneous nystagmus has completely subsided, **vestibular vertigo can no longer be present.**

Apparent contradictions to this latter generalization have proved something of a stumbling-block to those who have been able to recognize only one type of labyrinthine vertigo. For example, a colleague of mine mentioned to me that a patient, one of whose labyrinths he had removed several weeks previously, continued to suffer from vertigo. I had an opportunity of examining this patient and found that her vertigo was distinctly not of vestibular type. There are many similar cases and they furnish the basis for the writer's contention, viz., that in diffuse suppurative labyrinthitis there are two quite distinct forms of vertigo, viz., (1) **the vertigo of vestibular irritation** which characterizes the acute stage of the disease, and (2) **the vertigo of vestibular paralysis** which characterizes the latent or quiescent stage. It is the latter type which the writer wishes very briefly to discuss.

Orientation. It is clear that the power of maintaining one's equilibrium in all customary positions of the body depends upon what is called orientation, or the subconscious knowledge of the position of the body in space. If through accident, or attempting the unusual, the body is thrown into positions in which its usual relations to the three planes of space are reversed, orientation becomes defective or insufficient, and the subjective disturbance known as vertigo results. Again, if the faculty of orientation is suddenly disturbed or ablated, the individual at once loses his sense of stability in space, or in other words experiences vertigo. Obviously a study of this form of vertigo is a study of defective orientation.

That the static function of the labyrinth is the most important element of orientation, there can be no doubt. The vestibular organs cannot, however, be regarded as the essential organs of orientation, for after removal of one or both labyrinths, the individual regains in time his equilibrium. There must be, therefore, other contributory factors, and these are found in the muscular and arthrodial senses, the tactile sense and the sense of sight. Nor is it difficult to appreciate their importance to correct orientation, for

while it is the vestibular function to inform one as to the position of one's body in space, it is obvious that only through the tactile, muscular and arthrodial senses can one appreciate the relation of one's body to that on which it rests or is supported, and the positions which the various parts of the body occupy in relation to each other.

The investigations of R. T. Slinger and Sir Victor Horsley,* published in 1906, have so important a bearing upon orientation, and indirectly upon the vertigo and ataxia of vestibular paralysis, that we shall take time to discuss briefly their results. The purpose of these experiments was to measure the approximate accuracy or normal degrees of error in what they called topognosis, or "the orientation of points of space" by the muscular and arthrodial senses. These experiments were carried out in the following way:— The person experimented upon is blindfolded, and before him is held a glass plate graduated in squares of a half centimetre. The plate being placed in one of the three planes of space,—let us say the saggital-vertical,—i. e., a vertical plane bisecting his body antero-posteriorly,—the left hand is passively moved in different directions and the tip of its forefinger then brought in contact with some point upon the plate. He is then directed to bring the tip of the right forefinger to a corresponding point upon the opposite surface of the plate. It is obvious that in this test no information is gained through his tactile sense, which would convey the same impression wherever the finger might rest. Sight is eliminated by the blind-folding of the patient. Orientation must therefore be chiefly attributed to the muscular and arthrodial senses. This experiment was repeated with a large number of normal persons, the glass plate being held in different planes,—i. e., the saggital-vertical, the frontal-vertical and the horizontal. It was found that there is a physiological or normal variation or error which varied greatly according to the position of the plate in relation to the body. Thus, with the plate held in front of him in a

*R. T. Slinger and Sir Victor Horsley; "Orientation of Points of Space," etc. Brain, April, 1906.

vertical plane corresponding to the medial antero-posterior plane of the body, and at a height bringing the wrists to about the level of the lower end of the sternum, the average physiological variation or error was found to be 1¼ to 1½ centimetres, usually in the direction of propinquity,—i. e., the right forefinger being placed nearer the body by 1¼ to 1½ centimetres than the finger passively placed in contact with the plate. As the plate, still in the same vertical plane, is gradually elevated,—e. g., first to a level opposite the neck, then opposite the head and lastly above it,—the average error of orientation shows progressive and very rapid increase. When the experiment is repeated with the plate in the same plane but behind the person examined, the error is even greater. From these variations the investigators concluded that "there is a progressive diminution of knowledge of space as we pass outward from the central axis of the body."

These experiments were repeated with 22 adolescents or young adults who had become totally and permanently blind in early childhood, and with this interesting result,—viz., that the blind subjects exhibited similar errors, which also varied in accordance with the level or height at which the plate is held; but the errors were distinctly smaller, the average error being less by one-fourth than the average error of people with normal eyes. From this they concluded that "if the information gained by sight is permanently blotted out, the muscular sense under necessity can by education be brought to a point at least one-fourth better than that learned by the normal person."

The above experiments are particularly interesting and important in their bearing upon the compensatory power of one part of the complex mechanism of orientation to assume the work of another part whose function is annulled. We have seen that in the normal man orientation is a complex function in which the vestibular apparatus, the muscular, arthrodial and tactile senses and sight all take part. When sight is lost, muscular and arthrodial impressions become more sensitive and accurate. When one vestibular apparatus is destroyed the opposite labyrinth must become accus-

tomed to unopposed, unilateral activity, and the muscular, arthrodial and tactile impressions of space and position must be immensely increased. Until this process of involuntary education is well advanced, it is not surprising that the individual experiences occasional vertigo and ataxia, though he does not exhibit nystagmus and may show no constant tendency to fall in any given direction.

Clinical Manifestations of Vestibular Paralysis. We know that the onset of diffuse suppurative labyrinthitis, or the removal of one labyrinth, is invariably followed by very severe vertigo and disturbances of equilibrium,—these symptoms being the result of hyperactivity of the sound, unopposed labyrinth. In other words, sudden destruction or removal of one labyrinth is in effect equivalent to irritation of the remaining sound vestibular apparatus. After these symptoms of vestibular irritation have completely subsided, the patient is comparatively comfortable. He has now passed the stage when sudden movements of the head cause nystagmus and its attendant phenomena, and may be up and walking about his room. On getting out of bed in the morning, however, and on making any sudden and pronounced changes in the position of his body,—e. g., as in stooping over to pick up something from the floor, or even on suddenly rising from the sitting posture,—he becomes momentarily dizzy, and may even require support to prevent his falling. These symptoms are due solely to defective orientation, and though at first occurring frequently, are very slight as compared with the prolonged and distressing rotary vertigo of the acute stage. The attacks grow rapidly less pronounced, and may apparently cease within a few days, so that the patient is led to believe himself cured. He is likely, however, to experience a severe recurrence on attempting any physical act to which he has not re-accustomed himself. For example, when he first attempts to walk down an inclined plane,—e. g., in going down stairs,—he may experience sudden and severe vertigo, due to the sudden call upon his still defective faculty of orientation

by this new situation; and this may cause him to fall. Having had this experience, he calls into compensatory activity certain other senses,—chiefly the muscular and arthrodial senses and the sense of sight,—and soon learns to go up and down stairs without fear or danger. Again when he first attempts to walk in the dark,—e. g., in getting out of bed at night,—he may lose all sense of direction and may fall; and now must re-learn to maintain his equilibrium without the aid of sight.

All of the above manifestations of impaired orientation in the latent stage of suppurative labyrinthitis were demonstrated in the single case of a young physician on the house staff of the Manhattan Eye and Ear Hospital who was under the care of Dr. Duel. The same patient, having re-learned many of the common acts, such as stooping over, going up and down stairs, etc.,—was strolling along the street, and suddenly looked up at a tall building then in the process of erection. This deprived him of his sense of position or balance and he fell backward.

Even more typical of the vertigo of vestibular paralysis was a case which came under my care at the Manhattan Eye and Ear Hospital nearly two years ago. The patient, a man of 28 years, had contracted syphilis seven or eight months previously, and some five weeks before I first saw him, had become suddenly and absolutely deaf,—first in the left and then in the right ear.

Examination revealed the following condition: Both drum-membranes practically normal; absolute deafness of both ears, both by aerial and by bone conduction. Caloric reactions absolutely negative. The patient, seated upon a revolving chair, was rotated rapidly first in one direction and then in the other, with perfectly negative results,—i. e., there was no nystagmus, and the patient experienced absolutely no vertigo or discomfort. Clearly we had to deal with a patient both of whose labyrinths were absolutely non-irritable. Yet this patient complained as much of vertigo as of the deafness. Tests of his equilibrium demonstrated a type of ataxia presenting the following interesting con-

trasts with the ataxia of vestibular irritation: He walked with marked unsteadiness,—i. e., with a somewhat tottering gait, keeping his eyes persistently on the ground before him. Asked why he looked on the ground, he said "to keep from falling". Yet he was able to keep a straight course and walked wherever his will directed. With eyes closed and feet approximated (Romberg), he was able to stand steadily. In walking with eyes closed, his unsteadiness was greatly exaggerated, but he still contrived to maintain a fairly straight course.

As differentiating the ataxia in this case from that characteristic of vestibular irritation, this patient exhibited no nystagmus, complained of no sense of rotation, showed no tendency to fall in any given direction. He walked laboriously and with some difficulty, compelling his muscles to carry him where his will and sight directed.

The patient was admitted as a ward patient, where his symptoms were studied by some of my colleagues as well as myself. He never showed any signs of returning hearing, but his improvement in orientation was progressive and marked, so that when I last saw him, his gait presented no peculiarities which would attract the casual observer. In this he illustrated the rapid development of the muscular and arthrodial senses under the spur of necessity.

It sometimes happens that a case studied carefully at the time presents symptoms which are correctly interpreted only in the light of later experience. I mention by name such a patient, because her case was carefully studied by some of my hospital associates. Selena Miller, a girl then of 14 years, was admitted to the Manhattan Eye and Ear Hospital in November, 1908, with the following history: In July, four months previously, a radical operation had been performed on the right ear by a competent surgeon. The suppurative process had not been relieved thereby, and she had been re-admitted to the hospital on account of failing health and atypical symptoms of disturbed equilibrium. In the hospital she rapidly lost strength and flesh, and soon became rather apathetic and practically bed-ridden. When

induced to get out of bed, she could not walk without support. Support being withdrawn, she was apparently unable to maintain her equilibrium, and would fall. There was no constancy in the direction of her falling, and the character of her ataxia seemed so atypical that she was suspected of being a quasi-malingerer, and transferred to St. Luke's Hospital. After her retention there some days, she was returned to the Manhattan, no diagnosis having been made. On careful re-examination of the ears, it was found that the right ear was absolutely deaf, and gave absolutely no response to the caloric test. On exposing the old wound, I found a fistula leading into the horizontal canal and granulation protruding from the open oval window. Following an operation providing free drainage from the labyrinth, she made a fairly rapid recovery with relief of all ataxic symptoms.

The interesting feature in this case was the peculiar and excessive form of ataxia, which can be satisfactorily explained only on one hypothesis:— She had suffered suppurative destruction of one labyrinth; she was a delicate girl and the effect of a rather prolonged illness had been to reduce to an unusual and remarkable degree her muscular force and control. In the latter condition, her muscular and arthrodial senses were so far below par as to have failed utterly in compensating for the disturbed labyrinthine balance. Hence the patient's inability to stand or walk, and her liability to fall in any direction according to the chance position of the body. The disappearance of the ataxia cannot in this case be logically ascribed to the removal of the diseased and already non-functionating labyrinth, but rather to the relief of an extensive suppurative process, elimination of which enabled the patient to regain her muscular strength and tone, and her muscular and arthrodial senses to resume their rôle in the complex function of orientation.

The following case, which is the last I shall refer to, was in no wise unusual clinically. It will therefore serve the better to illustrate the point I wish to make, viz.,—the great importance of recognizing these defects of orientation and

their resulting phenomena as belonging logically among the late clinical manifestations of the disease.

L. M., 34 years of age, a carpenter, was admitted to the Manhattan Eye and Ear Hospital in May, 1910, suffering from chronic middle ear suppuration plus chronic suppurative labyrinthitis of the left ear. The labyrinth lesion was of long standing and the ear absolutely deaf and quite irresponsive to the caloric test. For this, a combined radical and labyrinth exenteration was performed. The immediate result of the operation proved that the static labyrinth was not wholly non-functionating, for he suffered a few days of characteristic vestibular symptoms,—i. e., rotary nystagmus, vertigo, etc. These quickly subsided, and the patient was soon up and about, and left the hospital to continue treatment in the dispensary. Some two months after the operation, while still under treatment for a slight discharge, the patient stated that he was in need of money and asked if he could take up his work as a carpenter, stating that he felt perfectly fit. I told him to do so, only cautioning him to make no arrangements which would prevent his coming regularly for treatment. The patient was a light-hearted, optimistic Italian, but when I next saw him he was deeply despondent. He told me that he had arranged to take up his work, but had been obliged to surrender it by the frequent attacks of dizziness which overcame him.

I had little difficulty in assuring him that his trouble was only temporary and would assuredly wear off in time. But while re-assuring him, I had time to consider the rather serious possibilities of such a case. Suppose that this man had been a builder's employee whose occupation required him to perform his work while standing upon a scaffold suspended high in the air. It is not difficult to imagine what might have resulted from an attack of vertigo for which he was wholly unprepared. Suppose that we are called upon to operate for diffuse labyrinthine suppuration upon a boy, and after all spontaneous symptoms have passed, allow him to resume the practice, dear to the heart of most boys, of diving into the river or swimming pool. May he not drown

before he can recover from the ataxia dependent upon a sudden disturbance of orientation?

The type of vertigo which I have endeavored to describe naturally shows some variation in different individuals. Ordinarily induced only by unusual demands upon the disabled mechanism of orientation, there are undoubtedly individuals whose muscular and arthrodial senses are far below the normal average of efficiency, and who after destructive labyrinthitis logically experience for a considerable period a mild grade of vertigo and ataxia of more or less constant type.

To recapitulate: The point I have tried to establish is that there are two distinct forms of labyrinthine vertigo and associated ataxia,—one the familiar **spontaneous type,** due to vestibular irritation and occurring only during the acute stage of suppurative labyrinthitis; and the other **not spontaneous,** not constant, not necessarily accompanied by nystagmus, characteristic only of the latent, or quiescent, stage of the disease, and induced solely by sudden and unexpected calls upon the lost or defective orientation sense, in the maintenance of which the intact vestibular organs are normally so important a factor.

Discussion.

Dr. William C. Braislin, of Brooklyn, N. Y., was pleased to hear Dr. Kerrison say the prognosis is good in functional cases; that had been his own universal experience. He had never had an opportunity to classify them with the degree of accuracy with which Dr. Kerrison had done in the paper, but clinically they bear resemblance to each other. He invariably tells these patients that they will get better. They may get better and then relapse, but they will eventually get well.

Dr. B. Alexander Randall, of Philadelphia, said there might be on the nerve trunk itself a perverted effect of nerve stimulus, and some such effects may occur after the terminal organ has been detroyed.

Dr. Norval H. Pierce, of Chicago, had reason to believe that a labyrinth which has apparently been destroyed by inflammatory process may regenerate, and after a time not only may respond to the caloric test but may exhibit symptoms of irritation. He cited a case in which, before radical operation for cholesteatoma, the caloric and rotation tests were negative. Sometime after that the patient returned with marked nystagmus, with the slow component toward the opposite side, with marked vertigo. He had seen several cases in which an apparently dead labyrinth regained its action and responded, after a time, to both caloric and rotation tests. He was not clear in his mind as to just how real vertigo could occur after a labyrinth had been destroyed and remains inactive, unless there is some regeneration of the deep muscular senses, pyramidal tract or something of that kind. It was well known that where a very large meatal opening is left after radical operation the patient becomes dizzy upon exposure to air. He cited a case in which a man was playing at golf in Chicago, when he fell down after his first tee, and another, a painter, who could not maintain his profession until the retro-auricular opening was closed, because he did not dare to go up the scaffolding. It was a somewhat new idea that one could have post-operative vertigo without, any sensory disturbance in the cerebellum.

Dr. Joseph A. Kenefick, of New York City, was interested in the subject of orientation in these cases. He did not understand whether the patient operated on at the Manhattan Eye and Ear Infirmary could still stoop over and maintain his equilibrium with visionary and vestibular functions both impaired. Before Barany's work was made known the speaker presented a case before the New York Otological Society, of a young woman who in childhood had been struck by a bullet on the right side, causing fracture of the skull, undoubtedly through the right vestibule, putting it out of commission. This patient, with closed eyes, immediately falls to the right side and has numerous scars

where she had fallen in the street. It would seem that Dr. Kerrison's case instead of growing better, would be more apt to grow worse. With any two of the three orientating senses out of commission it would be impossible to orientate. With any one of these impaired the remaining two may be developed to supply the needs of the patient's daily life.

Dr. Kerrison, in closing the discussion, said that Dr. Pierce had cited two very interesting cases, but that he hardly agreed with Dr. Pierce's interpretation of them. In the case of destructive labyrinthitis which after operation still showed typical vestibular symptoms, the nystagmus was clearly of vestibular origin—that is to say, it was due to over-activity of the remaining intact labyrinth which had not yet accommodated itself to a unilateral vestibular apparatus.

Dr. Kerrison said that he did not regard the two types of vertigo to which Dr. Braislin referred as identical. With vestibular vertigo there is always a tendency for the patient to fall in the direction opposite to the quick nystagmic movement, the direction of falling varying therefore with the position of the patient's head. The vertigo of vestibular paralysis, which Dr. Kerrison had described in his paper, is not necessarily accompanied by nystagmus and there is no constant tendency to fall in a given direction.

THE MORE EFFICIENT METHODS OF AURAL MASSAGE.

By B. ALEX. RANDALL, M. D., Philadelphia.

The theme chosen could hardly be less novel, not only because Simeon in the 11th century headed a long list of writers on the subject, but because late years have seen so many articles and inventions in this matter that now the appliances actuated by mouth-, hand-, hydraulic-, or electric-power are almost as numerous as the users or even as the patients submitted to them. It is because of this bewildering wealth of material and the diversity of the views expressed, that I shall essay to winnow out some of those points which have best met the needs of my experience. Others have doubtless as much right to enunciate conclusions differing from mine; but I believe that those who have not yet crystalized an experience of their own, will find my suggestions helpful.

Amidst the variety of apparatus, there is some risk that the principles may be lost sight of and the practice consist mainly in turning upon the patient some especially commended form of masseur. So it is well to recall that the ideal form of massage is that by the voice, even though few will undertake to use it rightly. Yet there are cases credibly reported in which an adult reared as a deaf-mute has had his vestiges of hearing so developed by a devoted wife's persistent use of her voice that it has become possible for him to hear and understand an unseen speaker in another room. But this is an electric age; and that man is regarded as quite fossilized who undertakes to use the voice for any such purpose except through the medium of some very up-to-date microphone. Twenty years ago a competitor for the Lanval Prize fairly proved that the microphone has no practical value for the deaf. But thousands are spent annually

in advertising the wonders wrought by such instruments, doubtless with a profit of tens of thousands to the advertisers.

It is too generally ignored that the benefit of the telephone of whatever form is in "transposing" the pitch of the conversational voice out of its ill-heard register into a higher key where the listener's hearing is little if at all impaired.

The magnification of the sound is usually a drawback, being of the sounds least desired; and it can be distinctly detrimental. The usually attenuated sounds transmitted by the telephone can be exhausting to the ear, if only because of the attention required to hear them amid any irrelevant sounds and because of the jarring noises sometimes intercurrent. The massage-value of such tones is minimal, since their pitch is often such as to allow them to pass directly through the drumhead to cause molecular vibrations at the fenestra, with little or no vibration of the chain of ossicles.

Much of what has been accomplished with massage-apparatus we owe to Siegle (Deutsche Klinik, 1864), whose pneumatic otoscope remains one of the best appliances at our command. A bulb excells his earlier method of giving the pressure and suction, although we are generally wise to use no more force than can be obtained by the mouth—i. e. probably about 5 pounds of positive and negative pressure. Under its use the response of the tympanic tissues can be in great measure seen both as to their mobility and their injection under such stimulation. The criticism has been advanced that all congestion is harmful and tends to further sclerotic changes. On the contrary, we have no other means so capable of obtaining absorption of pathological exudates. Whoever has studied in the eye the absorption wrought by Dionin or the clearing of corneal opacities under massage with ung. Hydragyri oxidi flav. can readily refute such a mistaken claim. That the massage can be abused only proves its potency, unless it can be shown that it cannot be employed without excessive action. Re-examination of the ear shortly after such congestive response demonstrates that

injection has soon disappeared; and the main value which I claim for my method of spraying Dionin **per catheter** is that its congestion of the tympanic mucosa serves to promote absorption of pseudo-anchylotic bands or thickenings. The pneumatic speculum should not be too deeply introduced, should not press too much on the veins of the front wall damming up the tympanic effluent (although some of this is advantageous, as in Politzer's plugging of the canal in the hope of causing absorption of the air outside as well as within the drumhead); but too marked a "passive hyperaemia" might cause extravasations. A bit of rubber tubing should usually be placed on the tip of the speculum to insure light air-tight closure of the meatus without bruising: the bulbous tip of the Delstanche and other instruments is rarely so good.

Some writers have so little grasped the principles of the use of the masseur as to deny that any appliance except some modification of their own, permits of suction. Any form of the instrument, simple or complex, can be made to give positive or negative pressure or both. Individual preference will modify the instrument in various details—for myself I much prefer the magnifying form as making quickly clear many details of the fundus which might escape even prolonged study of the unenlarged tissues. The pertinent fact is that valuable massage of the drumhead and ossicular chain can be thus accomplished. We are usually dealing with a depressed drumhead and wish to restore it to its normal tension and position. Suction rather than pressure is to be used; but all of our efforts to "suck outward" the drum-membrane avail nothing unless there is air in the tympanic cavity. Our lessened pressure without permits the expansion of what air is within the tympanum to force the drumhead outward. Failure to secure visible motion may not signify adhesions or rigidity—only an absence of air back of the tympanic membrane. Hence inflation by the Politzer method, or better as a rule by the catheter, may be a prerequisite to any mobilizing of the drumhead and the ossicular chain by pneumatic massage.

A perforation of the tympanic membrane may preclude any difference of pressure-change on its inside and outside, and our pneumatic massage then fails to elicit any visible motion. This does not prove that no motion is secured; on the contrary any fluid in the tympanum may be seen to move. The varying pressure upon the stapes and round-window membrane may excite marked motion with nystagmus, vertigo, and even syncope. The most striking examples of the "fistula symptom" are often cases with no history or sign of suppuration but presumably with a very relaxed stapedeo-fenestral articulation—light massage may bowl them from their seats more easily than violent syringing would do it for other patients. So the absence or immobility of the drumhead and ossicles does not mean that we can accomplish nothing useful by our massage. As before intimated, the mere cupping effect may by its congestion increase the plasticity of the parts with ulterior, if not immediate, advantage.

Since the effect of the pneumatic massage upon the broader surface of the drumhead may be far more potent upon the important factors of the conducting apparatus than that exercised through a perforation, it is well to remember that the open drum-membrane may be closed and perhaps instantly mobilized by the application of Blake's paper patch; and it is often for this special purpose that I employ this device. Conversely, it is often the pneumatic speculum which enables us best to adjust such patches and test the snugness of their application to the margins of the perforation which we wish to excite to reparative activity. I am a firm believer in the usual necessity of a mobile drum-membrane for the hearing of tones of low pitch; and as the rapidity of the vibrations of even our most rapid pneumatic masseurs is often far below that of the lowest audible tones, there is full need to avail ourselves of this rehabilitation of the impaired conducting apparatus.

The matter of the direct manual massage of the conducting structures of the middle-ear is too little dealt with by most authors. We know of Lucae's spring-probe; though

so little that one of Politzer's translators called it "Lucae's feathering-sound," and many were none the wiser or less so. Those who have seen or read of the device have generally been repelled and have made little or no use of it or its substitutes. Lucae himself gave up most of his mechanical additions and later preferred it in its simplest form; and this can well be further simplified by leaving off the spring. Any light probe, cotton-tipped, can serve the purpose. It is often notoriously painful as Lucae advised it: its tip was sometimes frozen to secure a benumbing of the short process or other parts touched. This is quite needless if we will cover its cotton tip with a salve—preferably of sticky Lanolin; then we can get both traction and pressure on the parts acted on, but so cushioned by the yielding of the buffer of salve that no spring is desired; and the light hand can impart to the precise region needing it a rapid and painless direct motion. And this has its best uses often after the destruction of the drumhead and larger ossicles, for we can work directly with the stapes and round-window membrane, stretching adhesions and effectually mobilizing conditions of pseudo-anchylosis. I have published cases of signal gain from this measure, as when in a lady of 30, whose useless right ear had been pronounced past all aid by one of our best men, the hearing was raised to hearing a forced whisper at five meters, and continued her sole dependence for the rest of her busy, useful life.

No small part of the efficacy of this direct massage lies in its inunction of medicaments capable of aiding the restoration and maintainence of mobility; so the tip of salve upon the probe can be adapted to the requirements of the case. The yellow oxide of Mercury has been my chief reliance, stimulant and absorbent in its action; but Ichthyol or its preferable alternate, Thigenol, has been much used in cases calling for more sedative effect. Not rarely in the chronic catarrhs, especially in more advanced life, I have been gratified by the good effect of Phosphorus oil 1%, commended as a help in "Presbykusis" by Sapolini. It may be wiped over the drumhead and walls and then gently

rubbed in by the massage as applied by the cotton-tipped probe to the short process, posterior fold and other regions. No "sweet oil" will surely remain sweet in the warm auditory canal, and so this application has in a few instances irritated sharply; or it may have been incompatible with some other medicament simultaneously used.

Many regard the hand-power appliances as obsolete and use and advocate those actuated by hydraulic, pneumatic or electric motors as the only up-to-date instruments. Some employ the massage for five, ten or even twenty minutes at a sitting and must save themselves this tedious manipulation —a practice which cannot be commended. Some regard the speed of vibration required for the best results to be beyond that obtainable by the hand. It is difficult with the Siegle actuated by hand-power to get ten motions per second, while the other devices may much surpass this speed; and it is argued that the lowest tones audible are all above 16 if not 32 per second. Still it is true that good results can be secured by the slower motions with perhaps greater safety than by the more rapid; and I believe that muscle-sense and brain are better controllers of the massage than any mechanical device.

On the other hand, to offset the value claimed for such automatic appliances as the electric masseurs must be placed the danger of rhythmic massage. We have long known that the bridge is in peril on which too many persons or cattle are moving in step; and the molecular changes which deteriorate metals and other dead materials still more endanger living tissues. A persistently vibrating tuning-fork among guinea pigs quickly induces cataract in their eyes (v. Stein). The dentist has had largely to abandon the use of the electric hammer which beautifully compacted the gold of his fillings but killed the nerve beneath. Similar vibrations will doubtless degenerate the cochleal nerve with its ganglion cells and terminal organ, although the experiments of Wittmaack and others are not conclusive. We often find catarrhally deaf patients with more nerve-involvement than could be expected from the age, the duration or the other

elements of the case; and the question "Have you been using an electric vibrator?" is usually affirmatively answered, sometimes with the declaration that there has been evident loss since its use. It is easy to collect many parallels in the occupational deafness of the boiler-maker and other workmen. The vibraphone and similar devices put forth a decade or more ago were advocated especially by the homeopaths, perhaps because the harm done by the telephone gave, by the "law of similars," promise of benefit to the deaf. Those who employed them found that they did indeed stir up torpid ears; but that they soon exhausted dangerously the nerve-power. They silenced tinnitus (in cases where they did not cause it); but often they so reduced perception that all objective as well as subjective noises were lost. These banjo-like machines have sunk into silence, to be replaced by simpler "buzzers," one of which (as I was assured by its medical advocate) had cured a deaf queen and received at her hands a medal of honor. The tuning-forks have been tried in this matter, especially in Paris, but as one of our colleagues has reported, with results not yet satisfactory. Most of such appliances, like the perpetual-motion machine, lack the "adjustment of one cog" to complete the perfect apparatus. Some measure of vibratory pneumo-massage as well as phono-massage has been claimed for many of these inventions, but careful testing has failed to substantiate any such action in those which I studied.

Hand-power cannot, try as we may, be dangerously rhythmic; and for any length of "seance" which has seemed to me reasonable, has not been over-taxed in giving this pneumo-massage; while the eye behind the Siegle speculum can measure the effect in motion and congestion and so may control the force and rapidity of the vibrations propagated through the conducting apparatus. The dosage may thus be determined with much accuracy; and the attention can be devoted to the ear under treatment instead of to the management of a complicated piece of mechanism.

Yet the evil influences that make for impairment of the hearing are almost ceaseless in their activity, while the

surgeon's measures of relief are occasional and often much interrupted. A massage that can be put into the hands of the patient may be of great value, if it be not dangerous. Such a measure was given us in the "tragus-presse" of Hommel; but I believe that I much improved upon it in the "Massage by the finger-tip" which I have long advocated (Philadelphia Polyclinic, Sept. 28, 1895). It can be well employed by the surgeon, the patient or any other, with no need for apparatus; and some twenty years of its use has never showed me any harm from its free employment. It should be dosed in accordance with the response of the ear to the pneumatic speculum and limited to such duration, force and frequency as has thus been seen to be expedient. Like all simple measures it is easily improved upon by beginners who have not yet learned it; but I have not found a better form than that which I first advised. The pulp of the (middle) finger, generally of both hands simultaneously, is introduced into the meatus, drawing the targus forward and by slight rotation pressed as deep as the dropped jaw will permit. Then gentle pumping movements in and out furnish a massage which is mainly by suction and can be safely continued from thirty to sixty seconds at varying rates of rapidity. It can be as slow as sixty per minute or less, or increased until as a mere tremor it is more than ten per second; while the degree of excursion can regulate the force as fully as in the most perfect mechanical masseur. This can be well alternated with similar pumping by the flat of the palm, stretched tight as it is laid in contact with the auricle and arched by slight flexion of the hand. This latter measure is so inconspicuous that the patient, as though smoothing the hair, can apply it unnoticed in the most public place and lift a load of deafness and tinnitus just at the moment when such help is most desired. It makes also the readiest means of relief in vertigo and faintness occurring under treatment. A half dozen such applications each day have seemed none too much, and have been most useful adjuvants to the periodic treatments by the surgeon;

and they can well be continued during the intermissions of other measures.

Of the more elaborate forms of massage of the Eustachian tubes by the bougie, simple and vibratory, I have little here to say except that they are dangerous in even the most expert hands; but I cannot sufficiently insist upon the value of the gargle as a gymnastic and massage of the tubes and pharynx. Medicaments are often poorly applied by gargling; but we have in **heat** one that can reach where the water itself does not reach and its constringent effect is equalled by few drugs at our command. A stroking massage of the neck between the ear and ramus of the jaw can make itself well felt by the Eustachian tube, but is rarely used to much advantage except in expert hands; while the wonder-working manipulations of the osteopath have never in my observation been availing except in extracting fees from the credulous.

In closing this plea for the recognition of the dangers of phono-massage as well as of all pneumatic or direct massage that is regular and rhythmic, I wish to insist that there is a **pro** and **con** to every measure that has here come under discussion; and in the hands of experts who give unflagging attention to avoiding their evils they are all capable of being used to the advantage of our patients. But there are devices of almost ridiculous simplicity that fully equal them in my opinion for all good use; while the safety and availability of the simple old Siegle speculum and the home use of hand-massage, gargling and such measures precludes that they shall ever become obsolete. Let us strive, each and all, to be up-to-date in our knowledge and utilization of all that is offered for the betterment of our patients; but in this as in all else that is admirable, may we all be "gentlemen of the old school."

Discussion.

Dr. Walter B. Johnson, of Paterson, N. J., said all who have been some time in practice have been through the list of the various forms of tympanic massage,—violin vibrator,

pneumo-massage, electrical apparatus, etc. Each one had, in turn, been adopted.

He had used pneumo-massage in preference to the electrical, having accomplished more with this method than with any of the other mechanical devices. A patient who has been deaf and long suffering from tinnitus is psychologically affected by new apparatus in any form.

Much of the primary benefit attributed to the various new methods is due to the psychological influence. He was not prepared to say that they do no good, but he thought that while they do not do enough good to warrant their continued use in the greater number of cases they could not be dispensed with as a part of the armamenturism of the otologist.

Dr. Sargent F. Snow, of Syracuse, N. Y., said massage by means of the Eustachian catheter, the force used being governed largely by the hand, and thus so regulated as not to be excessive, had proved efficient in his experience. He used mechanical massage only for diagnostic purposes. Interrupted beats of air give a very efficient method of producing motion in the chain of bones.

Dr. Norval H. Pierce, of Chicago, thought a careful differentiation should be made in cases in which massage is employed. In nerve deafness massage does no good and may do harm. In otosclerosis it does no good. But in adhesive processes of the ear, if carefully used, massage does good without doing harm. This applies to pneumatic massage. In cases of adhesions there are small or large areas of tympanic membrane which are atrophic, pneumatic massage should be employed with great care, because the membrane may be still further stretched, particularly if suction is used. Instead of pneumatic massage in these cases suction massage, such as Dr. Randall described, may be utilized. When the tympanic membrane is thickened and adherent to the promontory, pneumatic massage in conjunction with attempts to release it surgically, is advisable.

In other words, this means of treatment cannot be swept aside, but in selected cases is certainly as beneficial as Politzerization.

Dr. S. MacCuen Smith, of Philadelphia, called attention to neck gymnastics, which he had employed for a number of years. Whether this causes a certain amount of massage of the drum membrane by extension through the tubes he did not know, but the probabilities are that by producing better circulation of the neck, thus causing a better blood supply to the organ of hearing, the improvement is effected.

This is accomplished by moving the head with considerable force in various directions, one set of muscles working against their opponents. He had had considerable success with this method in connection with other means of treatment.

He asked Dr. Randall whether, in using the Siegel Otoscope, the bulb should not be compressed before it is introduced, so that the drumhead is first drawn out rather than forced in. He had seen this instrument used in the hands of inexperienced men, and the patients suffered from vertigo as the result of forcing the membrana tympani and ossicles inward.

Dr. Randall, in closing the discussion, earnestly commended massage as an adjuvant, nothing more. Catheter inflation, as suggested by Dr. Snow, will give improvement in the presence of a large perforation, the passing of the air causing a certain amount of massage of the bones. In cases of adhesive process, however, as suggested by Dr. Pierce, there is a good field for the employment of massage-measures of every sort. The one he depended upon is catheter inflation, to which he considered the others important adjuvants. The patient who feels worse immediately after catheterization will find that this entirely disappears through massage. Movements of the head as suggested by Dr. Smith, may accomplish this, or massage by means of Siegle speculum will give it more. More than five

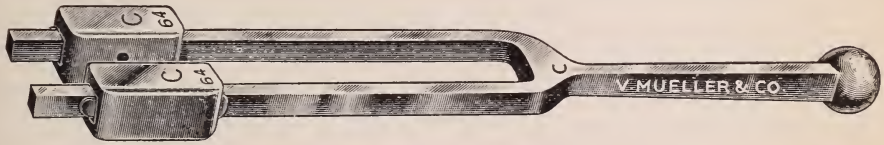
pounds pressure cannot be used by the head-movement, which is not sufficient. By means of the bulb which he uses fifteen pounds pressure per square inch can be used.

Referring to the neck gymnastics mentioned by Dr. Smith, it was perhaps worthy of note that in persons who are hung there is rupture of the drum membrane by outward pressure. This is an almost invariable finding. He wished to make it clear that the various simple massage measures are useful additions to other methods of treatment, and were warmly commended by him for this purpose.

A NEW AND SIMPLE METHOD OF SOUND EXCLUSION FOR THE PURPOSE OF DIAGNOSIS IN MALINGERING UNILATERAL DEAFNESS.

By NORVAL H. PIERCE, M. D., Chicago, Ill.

If a sounding body be fitted tightly into the external meatus, it has the power to exclude other sounds of greater volume. This result is due to the amplification of the sound waves in the external auditory canal by osseous vibration and to the obturation. Both Barany's and Neumann's apparati operate on these principles. If a tuning fork of



low pitch be tightly pressed into the external auditory meatus, like results are obtained. For this purpose we should select a fork of low pitch, because with such a fork we get a maximum of osseo-tympanal conduction. We should be guided as to the length of time the fork vibrates. As above a certain pitch osseous conduction diminishes and below a certain pitch duration shortens, it is best to select that fork giving both maximum osseo-tympanal conduction and having the longest duration. (A) Koenig is well adapted to this purpose, but any long prong A or C will give fairly serviceable results. It is very important to have the ends of the handle of the fork fit accurately into the external auditory canal. The spherical end on the Koenig rods secures this result acceptably. If the fork has a square handle, a conical or spherical end should be attached. For this purpose I have had made metallic spheres which may be

screwed on to the square ends of forks, such as are made by Reinhart or Gradenigo. A good deal of time was spent in experimenting with the best means of occluding the ear. Various substances were employed for this purpose, such as modeling wax rubber tubing. Dr. Sonnenschein, one of the assistants in my Clinic at the Illinois Eye and Ear Infirmary, fashioned a serviceable hard rubber plug which screws on the ends of the forks. But the spherical end serves the purpose best, and being made of metal can be readily sterilized.

In the cases of hysteria or malingering the method of procedure is as follows: The operator sits at the side of the patient facing the hearing ear, and asks the patient if he is sure that he hears well out of the good ear. The operator then strikes the fork against his knee, producing the maximum vibration, and the end of the fork is immediately thrust into the external auditory canal of the hearing ear, at the same time the auricle is drawn upwards, outwards and backwards, and then in a very subdued conventional voice the question is asked, "Do you hear the sound of the tuning fork?" In all cases of malingering and hysteria that have been tested up to the present time, the patient immediately replies in the affirmative. Or the patient is requested *while the fork is in position to* "say when the sound stops." Inasmuch as with the tuning fork vibrating in the well ear the sound of the voice was excluded from that ear the question must have been heard in the opposite ear, thus proving that the opposite ear is functionating for low conversational tone. The success of the procedure depends entirely upon the completeness with which external sound is excluded. In individuals with angular external auditory canals, it is best to have them open the mouth wide, so that the condyle of the mandible is thrown forward, thus facilitating the closure of the canal with the fork. In testing for hysteria, however, this maneuver had best be dispensed with, if possible, as the very simplicity of the procedure tends greatly to success. That sound is excluded by means of tuning forks tightly fitted into the auditory canals can be readily

proven by simultaneously stopping both ears with two tuning forks. If obturation is complete, we may use a voice which is considerably above the conversational voice without the slightest voice sound being perceived. Tuning forks, such as are usually carried around in our diagnostic case, have comparatively short duration. Therefore, during only the maximum amplitudes is sound excluded; consequently, only a few words can be used, such as "ten, happy, one," or such a question as, "Where do you live?" before the fork is again set in vibration. If lower forks are used, such as the Koenig $A—_1$ rod, we may occupy a much longer time in testing, in a much louder voice, before the forks are struck again. Care should be exercised not to stand in front of the patient where the operator's lips may be read, or too close behind the patient, in order to obviate osseous conduction.

Discussion.

Dr. B. Alexander Randall, of Philadelphia, thought the method of distinct value in malingering, especially in court cases. Its simplicity commends it.

Dr. Walter B. Johnson, of Paterson, N. J., thought the value of the method as a positive indication of the inability to hear is questionable, but as a means of detecting malingering it is of undoubted value. In the latter cases he believed failure practically impossible. It might, however, fail in persons who have received an injury, on a railroad or otherwise, in determining the loss of hearing with any degree of certainty. The conclusion might be reached that they were malingering, but the extent to which they can hear could not be determined. This is important because the question of damages depends upon the amount of injury to the hearing.

Dr. H. A. Alderton, of Brooklyn, N. Y., referred to a method of sound exclusion which he had found absolutely

effectual. A piece of rubber tubing, with terminal, is attached to the cut-off of an air tank; the terminal is pressed into the ear, and a certain amount of air-pressure released. This prevents the good ear from hearing, and, with the good ear completely excluded, the amount of hearing in the bad ear can be determined.

Dr. Ernest de Wolf Wales, of Indianapolis, Ind., called attention to the German method of sound exclusion by rattling a piece of paper in the good ear.

Dr. Pierce in closing the discussion, said the test depends upon the exclusion of air from the external auditory canals. It is not necessary to use paper; the hands rubbed over the ear will exclude sound. The method which he had just demonstrated could not take the place of Neumann's Method, but it could be used as a make-shift in the detection of bi-lateral deafness, and especially in the detection of malingering.

A SYMPTOM OF MASTOIDITIS.

By H. A. ALDERTON, M. D., Brooklyn, N. Y.

The average case of mastoiditis offers very little difficulty in the way of diagnosis; the indications being so evident that the merest tyro in otology recognizes the condition.

Beyond this region of certainty there is a middle field which is misty to the beginner or to the general practitioner, but which to the trained otologist is familiar ground. Some one symptom or group of symptoms, more or less obscure in themselves, indicating with considerable certainty the nature of the trouble and the necessity for intervention. Such symptoms, for instance, as drooping of the posterior superior wall of the external auditory canal in the neighborhood of the drum membrane; the persistence of a profuse, thick purulent discharge; the existence of a deeply congested and bulging drum membrane, notwithstanding extensive incision, associated with or without a profuse purulent discharge; the presence of a continuous throbbing tinnitus, which remains unabated after adequate incision and irrigation; the involvement of the deep cervical lymphatic glands anterior and posterior to the attachment of the sterno-cleido-mastoid muscle; the localized tenderness at the posterior edge of the base of the mastoid process over the site of the emergence of the mastoid emissary veins; the occurrence and continuance of a slight febrile movement, day after day; the depreciation in the general health, a slight or moderate malaise, accompanied by a coated tongue and odorous breath; the character of the infective agent, whether mild or virulent, the streptococcus, meningococcus, capsulatus and mucosus being especially of the latter type; the presence of a pulsating light reflex on the pus exuding from the perforation in the drum membrane; lastly, possibly, the evidence in the differential blood count of an in-

crease of polymorphonuclear percentage above 80 or an increasing percentage as contrasted with a lesser degree of increase in the leucocute count, this becoming more evident in successive examinations.

Finally there are a certain number of cases which puzzle even the most expert otologist. These cases may have only a slight discharge, which, however, keeps up unduly long considering the apparently moderate involvement of the middle ear structures; or the drum membrane may heal without returning quite to its usual normal appearance, the hearing remaining poor; there may be no evidence of a febrile movement nor any complaint of aching nor pain around the mastoid or in the depths of the ear, except at certain periods for a short time, especially at night; or there may be no tenderness on pressure or no confession of tenderness; no bulging of the drum membrane nor of the posterior-superior wall of the external auditory canal near the membrane. Sometimes there is complaint only of a dull ache over the affected side of the head. In these cases the throbbing tinnitus may and often is present but is not much emphasized, questioning at times being necessary to elicit the presence of the symptom. The general health may not seem to be affected. Frequently, however, after consent has been given to operation the patients will confess to a subconscious knowledge that something was wrong; often the patient confides to the house surgeon that he or she has no pain, feels all right and does not see any necessity for operation, only consenting because of confidence placed in the attending surgeon. And, occasionally, it is only on the appearance of external swelling that the otologist wakes up to the fact that the disease process has been steadily advancing during all this quiescent period.

Now it is just in these cases of doubtful diagnosis, though present as well, as a rule, in the other more evident cases, that the symptom to which the writer would like to draw attention is often present and of great value as corroborative evidence.

This symptom consists in a blurring of the outline of the mastoid tip as contrasted with that of the healthy side. The edges of the tip of the affected mastoid process are not so well defined as they are on the opposite side, or, when both sides are involved, as they should be normally. This blurring of outline is quite distinct from any localized swelling such as precedes a perforation of the mastoid cortex in the formation of a sub-periosteal abscess or a case of Bezold's mastoiditis. It may be the precursor of either of these conditions or it may occur when there is very little chance of either occurring.

The symptom is elicited by grasping the tip, anteriorly and posteriorly, between the fingers of one hand while exercising a control with the other hand on the healthy tip of the other side; the difference in definiteness of outline is then easily appreciable. The writer has had house surgeons and assistants, not knowing which was the involved ear, make out the affected side by this method alone. And in a large number of cases of acute mastoiditis, the presence of this symptom in conjunction with the existence of an acute middle ear suppuration has determined operation, though the patient was apparently progressing as favorably as usual; and in every case the operative findings have confirmed the value of the symptom as an important indication for operation. It goes without saying that a negative finding has not the importance attached the positive. The symptom is more difficult to elicit in fat or in very muscular people than in the thin and flabby. Of course, where both sides are affected the element of experience and tactile sensibility on the part of the observer comes especially into play and in these cases one may be misled more easily than in unilateral cases. While the swelling from otitis externa sometimes leads to doubt, this latter is almost always reduced in a few days by local treatment and the use of the ice-bag, upon which event the presence of the above mentioned symptom becomes manifest if an acute mastoiditis exists.

The blurring of the outline is, of course, due to a mild inflammatory infiltration of the periosteum of the mastoid tip and of the adjacent soft structures brought about by their juxtaposition to the underlying inflamed bone and seemed in most of the writer's cases to involve the entire periosteal lining of the mastoid process. The soft parts over the mastoid are more boggy than those over the unaffected side; this bogginess is at times sufficient to render the posterior auricular fold slightly less well defined in the affected side. But even where this bogginess is not apparent to the eye, the experienced hand will often detect the presence of the blurring of outline of the mastoid tip, the symptom on which the writer wishes particularly to lay stress, especially as occurring in cases of acute mastoiditis.

Discussion.

Dr. Lewis H. Taylor, of Wilkes-Barre, Pa., understood Dr. Alderton to say that this would be the determining point for operation although everything else is progressing favorably. Persons of very large experience might see some one thing to determine them to operate, but from his own experience he did not consider this symptom sufficient to warrant operation "if everything else is progressing favorably." He would not operate upon one symptom. He recalled a case in which he did not get to operate and in which the patient recovered.

Dr. Ernest de Wolf Wales, of Indianapolis, Ind., asked if it were not better to call this a sign rather than a symptom.

Dr. Walter B. Johnson, of Paterson, N. J., did not think Dr. Alderton had any idea of rushing into operation upon one symptom. He believed that a very large percentage of cases of otitis have inflammation of the mastoid cells in addition to other symptoms. A great many cases of this kind get well under the ordinary treatment for otitis. It is surprising, however, to note the amount of destruction which

can occur with comparatively very slight symptoms. He cited a case which had recently come under his observation in which the condition of otitis was complicated by furunculosis of the external auditory canal, and in which there was a succession of furuncles and some swelling of the neck below the mastoid. The question arose whether the involvement of the glands was due to the furunculosis or to periostitis resulting from mastoid inflammation. It was difficult to determine this point. The case progressed and finally operation was insisted upon, the patient not wishing it. The ordinary mastoid operation was proceeded with, whereupon it was discovered that while the external surface of the bone was apparently healthy, in chiseling the mastoid with the Killian Chisel the transverse incision seemed to separate the whole anterior aspect of the mastoid, the bone underneath being destroyed. This condition was connected with the diseased bone in the mastoid tip. There was no swelling over the mastoid, nor was there any distinct line between the swelling in the neck and that in the mastoid tip.

Dr. D. Harold Walker, of Boston, said the very slight swelling over the tip is one of the first signs to appear, and one of the last to disappear. A more important sign, in my opinion is the sagging of the posterior canal wall. If the canal wall remains perfectly round the prognosis is better than if it becomes more oval than round; in the latter case, operation is almost always indicated.

Dr. B. Alexander Randall, of Philadelphia, emphasized the difficulty which attaches to making a clean-cut differentiation in the case under discussion. It calls for extreme finesse in palpation. The index fingers are essential in learning all that is possible concerning these cases. He also emphasized the point that this is a sign of mastoid inflammation, but is by no means a positive indication for operation. Mastoid caries, is, of course, an indication for operation: but much inflammation or even empyema may occur, yet clear up without caries or pyemia.

Dr. Sargent F. Snow, of Syracuse, N. Y., depended largely upon the sagging of the posterior wall of the drum to determine the necessity for operation. The sign discussed in the paper should be of help but he would not operate upon that alone.

Dr. S. MacCuen Smith, of Philadelphia, asked Dr. Alderton to explain what he meant by "blurring". He also asked how soon, with the symptoms present, he would operate. He understood from the paper that the reader considered the presence of the symptom described sufficient to warrant operation, even in the absence of other symptoms. The drooping of the canal takes place only when the superior part of the cells are involved. There may be a serious mastoid conditon, necesitating operation, in which this part is not involved, but only the tip.

Dr. Alderton, in closing the discussion, refined blurring of the tip as a slight lack of definiteness with regard to the margins of the bone, whereas in the normal mastoid the outline of the bone is felt under the tips of the fingers, by which the margins can be limited. On the normal side the bone is hard and well-defined; on the affected side it is ill-defined, not boggy, but just not sharply outlined.

Replying to the question whether he would operate upon this symptom alone, he said he would not do that exactly, but if other symptoms were not well marked and this were present he would operate. He had done this and found good reason for operating. He cited the case of a Jewish woman who had no rise in temperature, and no indication that the mastoid was involved. The only symptom was that at night, about eleven o'clock, she had pain for a short time. At the end of four weeks swelling appeared over the mastoid. He then operated and found not only the mastoid but the sinus involved. The sinus operation was performed. The patient made a complete recovery, without any rise in temperature.

Answering Dr. Taylor's question, he said the more he had observed the symptom the more he had come to depend upon it as an indication for operation. There is quite a difference between this symptom and swelling. It is not a swelling in the sense that the bone is beginning to show indications that there will be perforation.

He did not agree with Dr. Walker that the sagging of the posterior canal wall is more indicative of operation than this symptom. It is always an indication for operation, but it is not as frequently present as this sign. In most of the cases of mastoiditis which come to operation this sign is present.

Dr. Randall suggested that this sign does not necessarily indicate operation. Its presence signifies that there is an underlying cause for trouble, and if there is enough trouble in the mastoid to give this sign he considered that it calls for operation.

CIRCULATORY AND RESPIRATORY REFLEXES AND NEUROSES FROM THE UPPER AIR TRACT.

W. SOHIER BRYANT, A. M., M. D., New York, N. Y.

The laboratory and clinical observations that have been made upon reflexes and reflex neuroses generally are now sufficiently well correlated to make it possible to demonstrate the vital importance of the upper air tract with respect to its reflex action. The upper air tract here considered includes all parts of the nose, together with the pharynx.

The term "reflex" is a loose one. It is commonly applied to all automatic acts operating in any parts of the nervous systems—the cerebrospinal and the somatic or sympathetic, notwithstanding the fundamental etiological differences in these nervous systems. Abnormal or pathological reflexes are usually called reflex neuroses. These latter are identical in their action with normal reflexes, the only difference being that the production of the reflex neurosis is abnormally easy, i. e., it occurs with a minimum, less than normal intensity of stimulation.

The distinguishing characteristic of the reflex is its conservative function. Its most definite purpose seems to be the protection of the organism against foreign and injurious elements. The cough, sneeze, hiccough, vomiting—these are typical protective reflexes arising in the upper air tract at the entrance of dust, smoke, food unfit for consumption, etc. The reflex mechanism guards the body against the intrusion of all harmful agents. Hence the importance of the upper air tract in its reflex action is obvious: it controls the intake of all nutriment—air, water, and food.

In the case of violent irritation of the upper air tract by smoke, noxious fumes or gases, a defensive attitude is instantly assumed; there is cessation of respiration during the expiratory movement, the purpose being to ward off or expel all harmful fumes. Conversely, in obstruction of the

upper air tract by foreign bodies, in choking, there is an arrest of respiration during inspiration. The aim here is to take in all the respirable air that can be obtained, so as to prevent asphyxiation. At the same time there is a backing up of blood in the pulmonary circulation, with consequent lung distention due to inhibited cardiac contraction. This phenomenon has a doubly conservative purpose—first, to increase the capacity of the lungs; and secondly, to reduce the supply of oxygen carried to the tissues, so as to defer complete asphyxiation.

These defensive manifestations of the organism are entirely non-pathological in their action. They are the response that every normal individual makes to harmful stimuli. Such reflex action can be considered pathological only when these reactions are produced with very slight stimulation, i. e., when abnormally profound reactions such as have been described are produced without violent irritation. We then have the reflex neurosis.

The chief differentiating characteristic of the reflex neurosis is its destructive action. As a typical neurosis of the upper air tract may be mentioned reflex asthma which concerns both the pulmonary and the circulatory systems, causing functional emphysema or pulmonary dilatation and pulmonary congestion. The mechanism of asthma is identical with that of the normal reflex when there is acute occlusion of the upper air passages. This causes dilatation of the lungs through impeded expiration and pulmonary blood stasis due to lowered intracardiac blood tension.

The reflex tendency to increase the lung capacity and the retention of respirable air is a conservative measure. The corresponding reflex neurosis—which is destructive, like all reflex neuroses—prevents the individual from using his otherwise comparatively normal respiratory mechanism.

In the reflex neurosis the exciting stimulus starts in the upper air tract and conveys the pathological centripetal impression through the trigeminus nerve and the sphenopalatine ganglion to their respective centers. The impression conveyed is the same as when the upper air tract is

seriously occluded. The centrifugal impulse passes out over the pneumogastric to the heart and the phrenic to the pulmonary apparatus, causing the typical condition of asthma.

For our knowledge of these reflexes we are especially indebted to Grossmann of Vienna, who in his experiments upon living animals has demonstrated that through stimulation of the upper air tract by artificial occlusion normal reflexes similar to asthmatic symptoms may arise. Experimental and clinical observations demonstrate that the asthmatic syndrome may appear in varying mild degrees. There may be slight functional emphysema or slightly impeded expiratory movements only, with moderate engorgement of the pulmonary circulation.

The writer's clinical observations have led him to realize the vital importance of the upper air tract in old age, and in chronic and acute disease when some of the asthmatic symptoms are present. Whole or partial occlusion of the upper air passages in such cases is of the utmost significance to the patient. Even slight contacts between the turbinal bodies of the nose and neighboring surfaces, or hypersensitivity of the mucous membranes without any contacts at all, may be causative of symptoms that in connection with other pathological conditions will produce fatal results. I believe that not infrequently the reflexes of the upper air tract determine whether or not the other pathological conditions will terminate fatally. Through their inhibitory cardiac action and interference with respiration, these reflexes in many cases augment the gravity of disease to a material extent.

Etiology of the asthmatic reflex and asthmatic neurosis. As we have seen, the asthmatic reflex is caused by obstruction of the upper air tract. It is the unconscious expression of nasal stoppage and air hunger. The asthmatic neurosis, or true asthma, is due to hyperesthesia of the mucous membrane of the upper air passages and to a deficiency of inhibitory power in the nerve centers. The deficiency in the nerve centers is usually hereditary, or it may be acquired through exhaustion or toxic irritation. The hyperesthesia

of the mucous membrane appears also to depend upon heredity, sensory exhaustion, or overstimulation and toxic irritation. Local causes such as toxic irritation and morphological irritants, as well as irritation from impure air, may be the cause of the hypersensitivity of the mucous membrane. Asthma is the unconscious expression of the neurotic impression of nasal stoppage and air hunger.

Diagnosis. The diagnosis of these obstructive and hyperesthetic conditions can be demonstrated in two ways:

(1) By shrinking the engorged tissues with adrenalin to a sufficient degree to open the passages, thus relieving the local surfaces from irritating contact and affecting amelioration of the asthmatic symptoms.

(2) By anesthetizing with cocaine the nerve terminations that supply the constricted region or the hyperesthetic membrane.

If either of these methods produces marked amelioration of the pulmonary and circulatory symptoms, the diagnosis of the asthmatic reflex or neurosis is justified.

Treatment. The emergency treatment of these asthmatic cases is limited to local applications. In chronic conditions where time will permit, the nasal contacts, obstructions, and hyperesthetic areas should be removed by the usual therapeutic and surgical methods.

Prognosis. The results to be anticipated from treatment are improvement of the circulatory and respiratory symptoms—in other words, a profound amelioration of the pathological conditions. Complete recovery seems to depend upon continued treatment of the upper air passages. Through my clinical experience I have been led to expect that in urgent cases, local applications will afford instant relief varying in length of time from one hour to several hours; and that if life is prolonged and treatment continued, the fundamental pulmonary and circulatory conditions tend to recover. The intervals of relief will lengthen rapidly in response to repeated applications.

who do not live up to the ordinary hygienic rules, perhaps, in the matter of tobacco, alcohol, exercise, etc., and who suffer from autointoxication from intestinal fermentation. All these are causes of congestion in the turbinates.

Dr. S. MacCuen Smith, of Philadelphia, agreed with Dr. Richardson. Anything which causes an intestinal upset causes a reflex conditon in the nose. Personal experience substantiated this view. He had had an acute nasal catarrh with congestion, which lasted two weeks. At the end of the two weeks he developed a very irregular heart. He then became alarmed and consulted a specialist, who assured him that he had no organic trouble with the heart. The heart trouble went on for two weeks more, at the end of which time he developed a violent diarrhea. A fellow member of a social club suggested that he take a spoonful of bicarbonate of soda in a glassful of water. He did so, and the diarrhea ceased, as did also the heart trouble.

Dr. Bryant, in closing the discussion, stated that the question of the etiology of reflex neuroses from the upper air tract is irrelevant to his paper which concerns itself with a consideration of these phenomena regardless of their origin. Dr. Smith's experience, he stated, emphasized the points of his own paper.

REMARKS ON THE DIFFERENTIAL DIAGNOSIS OF SEROUS AND SEPTIC MENINGITIS.

By NORVAL H. PIERCE, M. D., Chicago, Ill.

The differential diagnosis between serous meningitis and septic meningitis is occasionally accomplished with difficulty, and the differential diagnosis between localized purulent meningitis and diffuse meningitis is often impossible. The cause of this is that some of the symptoms of a purulent meningitis are caused by the mere increase in the cerebrospinal fluid as it occurs in pure serous meningitis, such as headache, stiff neck, choked disk, Kernig's sign. In cases of pure serous meningitis, such as Quincke in his first papers alluded to, and in which the symptoms were ascribed to the increase in the cerebrospinal fluid, there may be little or no temperature, but in serous meningitis occurring in the course of an ear suppuration this factor of temperature may be added by the primary disease. For instance, in children and infants, we occasionally observe all the symptoms of septic meningitis together with fever which immediately disappear after spontaneous or surgical perforation of the tympanic membrane, or after a lumbar puncture. The fever in these cases is not due to the increase in the cerebrospinal fluid, but to the inflammatory processes going on in the middle ear. In cases in which there are symptoms of meningitis accompanying thrombosis of the sigmoid sinus in adults, which disappear immediately on freeing the septic contents of the sinus, we have the usual septic temperature, but this temperature again is referable not to the processes going on within the dura, but to the septic distribution from the primary clot. Increase of the cerebrospinal fluid can scarcely be regarded as an inflammatory phenomenon in the sense that we regard the changes taking place in the pia in septic meningitis. The antipodal clinical courses precludes any

Illustrative Cases.

(1) A man forty years old. Acute infection of the respiratory tract, "grippe". Face white and haggard. Nasal breathing. Pulse intermitting many beats; almost stopping. Respiration labored and shallow. Temperature 102 degrees. Sensation of suffocation and imminent death. Patient apparently **in extremis.** Foot of bed raised. Subcutaneous injection of strychnine had given no relief. Applications of cocaine and adrenalin along lower and middle turbinals brought instant relief from subjective symptoms with improvement of respiration and regular pulse. Relief lasted four hours, when there was another attack of dyspnea, palor, and heart stoppage. Another nasal application relieved the condition more completely, after which the patient passed to final recovery in a few days.

(2) A man seventy-three years old. Recent fracture of arm from collision with automobile. Pale, heavy, stout man, of senile appearance, nearly bedridden and despondent. Before accident was fairly active and comfortable. Pulse slow, weak. Respiration slow. Nose breather. Nasal inspection showed hypertrophied turbinals, no notable respiratory obstructions, but contacts. Repeated shrinkage of the hypertrophic turbinals by adrenalin and iron salts soon relieved contacts in nose. The circulation and respiration improved and patient was restored to as good or better health than before accident. In two months the patient was looking and feeling very well and much younger.

Conclusion. In conclusion I wish to say that unless the proper air tract is in no way obstructed and is not hyperesthetic—which is rarely the case—the patient suffering from emphysematous symptoms and pulmonary congestion is not receiving the attention to which he is entitled if the upper air passages are ignored. Under such circumstances we cannot over-emphasize the importance of treating the upper air tract, so as to minimize the danger arising from the reflex neuroses which are so abundantly found to emanate from this region.

Discussion.

Dr. Joseph A. Kenefick, of New York City, expressed his personal indebtedness to Dr. Bryant for his scientific analysis of the condition with which specialists are constantly confronted, particularly with reference to "nasal insufficiency." Old persons whose turbinals become engorged, so that when they lie down at night the nose is completely shut off, suffer from a row of complications just described and due to nasal insufficiency. In these old people astringent medication to the turbinals is beneficial, converting them into normal breathers, and adding years to their lives. Dr. Bryant had cleared up some of the obscure scientific principles associated with this condition.

Dr. B. Alexander Randall, of Philadelphia, said those who had studied with Stillè were taught that in the treatment of catarrhal congestions of the upper air-passages no single medicine is so valuable as the simple mild chlorid of mercury. Calomel, as a dusting powder for these parts is slowly absorbed, especially in the presence of oil, acting as an astringent and alterative. So long as it is not absorbed it is an excellent protective. Some of it is converted into the bichlorid and so exercises local antiseptic influence. It is an admirable measure in such cases as this, which will be made worse, perhaps, by reaction after adrenalin and cocain. It is possible by this means to hold the gain made by the more positive shrinkage with cocain, so valuable for diagnosis and access.

Dr. Charles W. Richardson, of Washington, D. C., thought the whole question resolved itself into whether these reflexes are local in origin in the upper air tract, or whether they are of general origin. Whether, in other words, changes in the nerves of the nasal portions of the upper respiratory tract are not secondary to those of general origin. In the cases mentioned by Dr. Kenefick the condition is of general rather than of local origin in that they occur in old people

who do not live up to the ordinary hygienic rules, perhaps, in the matter of tobacco, alcohol, exercise, etc., and who suffer from autointoxication from intestinal fermentation. All these are causes of congestion in the turbinates.

Dr. S. MacCuen Smith, of Philadelphia, agreed with Dr. Richardson. Anything which causes an intestinal upset causes a reflex conditon in the nose. Personal experience substantiated this view. He had had an acute nasal catarrh with congestion, which lasted two weeks. At the end of the two weeks he developed a very irregular heart. He then became alarmed and consulted a specialist, who assured him that he had no organic trouble with the heart. The heart trouble went on for two weeks more, at the end of which time he developed a violent diarrhea. A fellow member of a social club suggested that he take a spoonful of bicarbonate of soda in a glassful of water. He did so, and the diarrhea ceased, as did also the heart trouble.

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such view. The increase in the fluid must be regarded rather as a secretory phenomenon than as an exudate or transudate, and is caused in all probability by the action of toxic substances introduced from a foreign inflammatory focus without the presence of living organisms. A walled-off meningeal focus may be regarded as foreign in this sense. The difference between the clinical course exhibited by a serous meningitis, or certain forms of localized septic meningitis, and that presented by diffuse septic meningitis, to my mind is sufficient to differentiate them clearly one from another in a pathological sense. We have all seen cases with meningeal symptoms in the course of an acute otitis media, or an acute exacerbation of a chronic suppurative inflammation of the middle ear, which despite lumbar puncture, meningeal drainage, through and through drainage, or whatever other surgical procedure is selected, have been swept away in a few days to death, while in the case of serous meningitis, or a localized septic meningitis, as soon as lumbar puncture has been performed or the dura incised immediately covering the inflamed area, recovery begins. The difference between these forms of meningitis, as regards their clinical course, is due to the portion of meninges involved. Serous meningitis kills from brain pressure. In internal meningitis or internal hydrocephalus, which continues for some length of time, we have pressure atrophy of the brain due to the accumulation of fluid within the ventricle, or we may have death result from serous meningitis from pressure exerted by the fluid on the medulla oblongata against the foramen magnum, but death results in septic meningitis not from sepsis, but from destruction of the pia and with it the cortex. The pia invests all portions of the surface of the brain, dipping down between the sulci and carrying with it blood vessels, which penetrate its substance and supply its nutrition. Accompanying these vessels are peri-lymphatic spaces, and when the pia is involved these vessels together with the peri-lymphatic spaces are also involved and the septic process, so that death results from an encephalitis rather than from a pure meningitis. It is conceivable that

we may have an inflammation limited to either the subdural space or the subarachnoid space, and if this is so, it remains for the future to differentiate clinically between these two types. There are reasons for believing that such a differentiation may be made by that most trustworthy of differential diagnostic aids, lumbar puncture, and it is for the purpose of casting some light on this interesting field that I report the following cases:

CASE I. J. H., aged forty-seven, police officer, was admitted to the Illinois Charitable Eye and Ear Infirmary October 10, 1910. as an outdoor patient. He complained of headache, sleeplessness, and restless anxiety. For years there had been a discharge on the right side of the nose. Four weeks before admission to the infirmary, swelling and pain and redness were noticed in both frontal regions, more marked on the right side. At the time he was first examined there was edema of the right eyelid, swelling and redness and tenderness over the frontal regions, and root of the nose. Rhinoscopy disclosed a purulent secretion coming down from the anterior portion of the middle meatus, polypi, and polypoid degeneration of the middle turbinated body. He was advised to remain in the hospital, but refused to do so. On the twelfth he was brought to the hospital by his son. Headache had increased, and he had periods resembling coma. Eyes and head deviated to the right; positive Kernig's sign; motor paresis of the left arm and leg; vertigo; could not close the left eye; temperature 100.2; pulse 92; respirations 30. A double Killian was performed. The right frontal process of the superior maxilla was necrotic and could be removed with the dressing forceps. The ethmoid labyrinth came away as sequestrum; the left sphenoid sinus was full of pus, its anterior wall softened. The partition between the two frontal sinuses had already disappeared, but the right ethmoid cells were but very slightly affected. Lumbar puncture provided a fluid normal in color, consistency and tension. The patient's condition, far from improving after the operation, became worse. Rigidity of the

neck became more pronounced; there was fibrillary twitching of the muscles of the face; paralysis of the left arm and leg became more marked; coma continuous, so that on the fifteenth of October the inner wall of the right frontal sinus was removed. Clear cerebrospinal fluid escaped, and on probing beneath the dura no pus was discovered. Lumbar puncture a few hours before death disclosed perfectly clear cerebrospinal fluid, normal in color, consistency and tension. Patient died on the seventeenth. Post-mortem of the head. The skull was exceedingly thin in the temporal region, especially on the right side. On incising the dura a quantity of pus escaped. The pia over the entire right hemisphere was infiltrated and thickened and lymphatic spaces outlined in pus with free pus between the sulci, especially in the fissure of Rolando, where a microscopic destruction on the surface of the brain, and the cortex was discernible. The point of origin of the septic meningitis appeared to be from a focus of softening in the region of the right sphenoid cavity. Inoculations were made from the cerebrospinal fluid on gelatin, and serum glucose. After three days cultures were negative. Microscopic examination showed polymorphonuclear cells, mononuclear, and a few lymph cells. The percentage was not obtained.

1. Fissue filled with pus.
2. Thickened arachnoid-plastic material.
3. Adherent dura peeled back.
4. Infiltrated pia and ulceration of cortex.

CASE II. B. W., admitted to the Michael Reese Hospital, October 10, 1910. Patient complained of severe headache, with great physical depression, which had existed for five or six days. The onset of the headache was rather sudden, and he had vomited several times. Patient thought he had fever from the beginning of his trouble, and felt very cold once or twice. He gave no history of discharge from the ears.

Physical Examination: General appearance is that of a fairly well nourished man, lying in bed with head retracted, legs and thighs flexed, groaning with pain, which is referred to the frontal region. During the few hours he has been in the hospital he has had projectile vomiting. Examination of the left ear by means of a pledget of cotton showed that there was a slight amount of very foul secretion. Otoscopy revealed cicatricial adhesions bringing the malleus to the promontory with a fistula in the posterior superior quadrant. No tenderness or swelling over the mastoid. Right pupil smaller than the left, both react to light and accommodation. No spontaneous nystagmus. Caloric reaction negative. Pupils were dilated with two per cent. homatropin solution; at the center of both corneae small opacities. Examination of the fundus shows absence of neuritis. Both discs on temporal side show myopic choroidal changes. Veins are fine but not tortuous. External strabismus of right eye; no herpes; tongue is dry and coated; breath foul; examination of the nose negative; marked rigidity of the neck and tenderness over the spine. Thorax negative. Abdomen thin, soft, no tenderness, retraction or rigidity, no hepatic or splenic enlargement. Knees and thighs are flexed; Kernig's sign positive; knee jerks brisk; ankle clonus more marked on the left than on the right side. No Babinski; hyperesthesia of the skin over the extremities.

He lay in a semi-comatose condition from which he could be aroused, when he would only moan and complain of intense headache. As he spoke little or no English, it was difficult to ascertain the condition of his speech centers, but the probabilities are that aside from the slow cerebration these were not affected.

On October 13 I performed a radical mastoid operation. The sigmoid sinus was exposed, and found healthy; the tegmen tympani et antri very much discolored and softened to the extent of a cubic centimeter. The bone was easily removed with dressing forceps, disclosing a discolored and thickened dura, with no normal brain pulsation. The dura was incised cricially; adhesion between the dura and the surface of the brain, but no cerebrospinal fluid escaped. Exploratory puncture upwards into the brain substance for about one and a half cubic centimeters, the grooved director entered an abscess containing about four centimeters of foul smelling pus. A glass tube bent to fit the mastoid excavation was introduced into the abscess cavity and the patient returned to the ward. The next day the patient's sensorium was clear; headache practically had disappeared, and he made a rapid and uneventful recovery. Patient's temperature at the time he was admitted was 102.6; pulse 80; respirations 29, which ran up to 103.2, and pulse was 72, respirations 28. After operation temperature was 99.6; pulse 80. From that time until it sank to normal, three days after the operation, it did not go above 100.4; pulse 76.

The cerebrospinal fluid, which was obtained on October 12, was opaque, resembling thin pea soup. Direct smear showed many pus cells, but no organisms. Differential count showed small mononuclears three per cent.; large mononuclears eight per cent., and poly-morphonuclears eighty-nine per cent. Cultures from the cerebrospinal fluid remain sterile after 48 hours. Pus from the mastoid showed the staphylococcus and a Gram negative motile bacillus, probably the colon bacillus. Urinary examination negative.

How are we to explain the peculiarities in these two specimens of cerebrospinal fluid? In the one case we had an extensive septic meningitis, as proved by post-mortem, with clear cerebrospinal fluid, under no tension, and on microscopic examination showing few cellular elements.

In the other case, the course of which precluded a belief in the existence of anything but a small localized area of meningeal inflammation with increased cerebrospinal fluid,

the cerebrospinal fluid was opaque and rich in pus cells. It proves at once that the cytologic content of the cerebrospinal fluid cannot be depended on as differentiating between localized and general septic meningitis, or meningitis serosa. The one fact that is of great importance is that in the second case bacteriological cultures remained sterile. While from the first case the cultures remained sterile, I am inclined to place no reliance on these findings as the bacteriological manipulations were not carried on by a sufficiently expert individual. In other words, I believe that the only differential point that we can obtain clinically between a septic meningitis that is at the moment spreading or has become general, on the one hand, and, on the other, a localized meningitis that is effectually walled off and serous forms is the presence of living organisms in the cerebrospinal fluid, and this I base on a considerable experience. We must remember in accounting for the presence of the enormous cellular content in the first case that these bodies have a tendency to sink down and accumulate at the bottom of the cerebrospinal sac as they would in a test tube, so that a localized meningitis of small area, walled off from the general cerebrospinal cavity, may produce at the bottom of the cerebrospinal sac an accumulation of corpuscles, causing the fluid to be opaque, while the fluid in the ventricle may be quite clear.

It is exceedingly difficult to explain the absence of corpuscular elements. However, in spinal puncture we obtained the cerebrospinal fluid from the subdural space, and I will venture to remind you that within the skull the subdural space is only a potential space, and the subarachnoid space is a positive space; while in the spinal canal the subdural space is a positive space, and the subarachnoid space is a potential space. The only reasonable explanation as to the occurrence of clear cerebrospinal fluid obtained by lumbar puncture in the first case is that the subarachnoid space was completely and effectually walled off by the inflammatory plastic zones from the subdural space.

Discussion.

Dr. Ewing W. Day, of Pittsburg, Pa., called attention to the fact that one is handcapped by ignorance of the circulation of the cerebro-spinal fluid. Last winter he had tried some experiments along this line. He had a patient who was apparently suffering from meningitis. The fluid was sterile. He went in and tapped the right ventricle, the fluid from which was distinctly colored and filled with pus cells and micro-organisms. Five or six days later the cerebro-spinal fluid showed micro-organisms. He thought there must be some obstruction to the spinal fluid, that the small foramena leading from the ventricle to the canal must be closed. In another case the fluid was distinctly purulent. The patient died, and autopsy showed that there was no meningitis or signs of it, but that the patient had a brain abscess which had ruptured into the ventricle, causing the purulent condition of the fluid.

Dr. Day added that the ventricle may be completely collapsed, thus accounting for failure to get fluid through the canula, as in a case upon which he had operated. At autopsy in this case, the ventricle on the side upon which he went in was totally collapsed, and the ventricle on the other side had no connection whatever with this.

Dr. Walter B. Johnson, of Paterson, N. J., asked if he understood Dr. Pierce to say that the last case, in which there was recovery, and in which meningococci were found in the cerebro-spinal fluid, was not one of lepto-meningitis. It seemed to him that the negative findings might be considered reasonably positive evidence of the absence of lepto-meningitis or extension of brain abscess to the ventricles, or some other form of disturbance which introduces micro-organisms into the spinal fluid. The presence of micro-organisms, as evidenced by bacteriological examination, would seem to indicate that an inflammatory condition

existed in the cerebral cavity. Cases of infection of the cerebral cavity have recovered, and he would think this such a case.

Dr. S. MacCuen Smith, of Philadelphia, was interested in Dr. Day's explanation of why micro-organisms are not found in the cerebro-spinal fluid. The findings in the fluid do not help very much in the diagnosis. He cited a case which resulted fatally, in which the cerebro-spinal fluid was examined at regular intervals for a period of from ten days to two weeks. The fluid was always perfectly clear, and yet the patient died of a diffuse meningitis.

He asked Dr. Pierce if he had any means of definite localization of a brain abscess prior to operation; in other words, did he have means, from a neurological standpoint, of knowing just where this lesion was before exploration of the region selected? Also whether, in view of the fact that the patient recovered, did he feel that there might have been any satellite abscess formation, and if there had been such formations, would the patient have recovered? In his own experience, a fatal termination is usually due to satellite abscess formations.

Dr. James F. McKernon, of New York City, referring to the matter of the contamination of the blood by staphylococci, mentioned two sinus cases observed recently by him in each of which two cultures were taken. The first culture, after thirty-four hours, was negative; the second culture, after forty-eight hours, was positive. The symptoms were indefinite from clinical evidence, but he operated upon the combined evidence, and found a distinctly formed, organized clot 1⅓ inches in length. This was evacuated and the patient made a good recovery. It cannot always be said that staphylococcus contamination is due to skin infection.

The use of urotropin in cases of purulent meningitis was suggested by Dr. Cushing, of Johns Hopkins, who said it was impossible for steptococci to live in a concentrated solution of urotropin. Last winter, in practically hopeless cases after

everything else had been done, the speaker tried this agent. He drew off as many as eighty-four cubic centimetres of cerebro-spinal fluid, which was distinctly under pressure, flaky, and showing, under laboratory examination, pus and large numbers of streptococci.

This fluid gave a culture of streptococci. The canula was left in place in the cerebro-spinal canal, and through it he injected forty to fifty cubic centimetres of a concentrated solution of urotropin, the quantity of fluid introduced being about one-third less than that withdrawn from the cerebro-spinal canal. He treated four cases by this method, with three recoveries. One of these three cases was a boy, sixteen years of age, upon whom he performed the mastoid operation. At the time of operation perforation of the tegmen antri, a large peri-sinus abscess, and a large epidural abscess on the floor of the middle fossa, were found. Forty-eight hours after operation the patient was absolutely unconscious, and presented every symptom of meningitis—the eye symptoms, the reflexes, muttering delirium, and temperature of 106.2°, not of sudden but of general rise. He drew off seventy cubic centimetres of cerebro-spinal fluid and injected through the canula fifty grains of urotrophin. In less than six hours the temperature fell to 103°, within eight hours the patient could be aroused and would answer questions. He injected fifty grains more, and the next day the patient gave evidence of marked improvement, subsequently made a perfect recovery.

He proposed, in cases which come under his observation in future, not to wait until the last moment before giving urotropin.

Three cases out of four of purulent meningitis subsided without drainage of ventricles or canal.

Dr. Pierce, in closing the discussion, said there were no localizing symptoms. The clinical picture was that of meningitis. There were no pareses,—no thing that would localize the abcess. He always removes black bone. Beneath this, in the case cited, he saw discolored dura, and this he

always incises. In other words, he was led on by the findings. These are the cases which recover. The abscess in this case was on the left side. If micro-organisms are found in the cerebro-spinal fluid as suggested by Dr. Day, it indicates meningitis, but the presence of micro-organisms and pus cells in the fluid does not mean that it is general meningitis. It does not differentiate between general and localized meningitis,—"a consummation devoutly to be wished." The pia dips down into the sulci and carries with it blood vessels, around which are perilymphatic sheaths, and it is the infection of these that causes death from encephalitis. However, the bacteriological examination may turn out, it is of very little importance in differential diagnosis. If the disease is septic general meningitis the patient dies before a conclusive report comes from the laboratory.

The circulation of the cerebro-spinal fluid is exceedingly interesting, and by no means thoroughly understood. The subdural space in the brain is only a potential space, whereas in the spinal column it is an actual and large space. The subarachnoid space in the brain is an actual space, for a large part at least, and in the spinal cord it is a potential space. It would be impossible, with any technic at hand, to differentiate in spinal puncture between the subdural and the subarachnoid space. It is quite possible theoretically that there may be suppuration in one space, and not in another. One space may be blocked off from every other space. This may account for the confusing results from some part of the spinal puncture.

THE SIGMOID SINUS AND THE JUGULAR BULB IN INFANCY.

By WILLIAM C. BRAISLIN, M. D., Brooklyn, N. Y.

Surgeon Brooklyn Eye and Ear Hospital.

A structural weakness of the parts of the skull immediately adjacent to the ear, with its consequent lessened resistance to the invasion of infections, is a characteristic of early infancy. In infants, there is also a greater accessibility of the parts of the ear and surrounding structures to operative procedures for their relief.

In infants up to the age of five years or more, the common occurrence of edema and subperiosteal mastoid abscesses in a large proportion of cases of mastoid inflammation is due to the superficial situation of the mastoid antrum in these young subjects.

The more ready accessibility of the ear structures in infants also applies to the sigmoid portion of the lateral sinus and the jugular bulb, partly by reason of the lesser density of the bone covering it.

Thus the jugular bulb of the new-born is of much less actual depth due to the small size of the cranium. It is also worth notice that its intra cranial opening (the jugular foramen or foramen lacerum posterior,) is really more exteriorly situated, relatively, than in the adult. The sigmoid portion of the lateral sinus, as it passes downward and inward across the temporal and occipital bones, proceeds a relatively less distance, in the infant, before passing forward into the jugular bulb. The passage of the vessel formed by the junction of the lateral and inferior petrosal sinuses, from within the cranium outward is quite directly forward and slightly downward and lacks the tortuosity and much of the bulb-like enlargement of the same structure in adult life.

The intracranial opening of the jugular foramen in the infant is situated about midway between the rim of the foramen magnum and the periphery of the cerebellar fossa (about ½ inch on either direction), and externally at a like relative position; while in the adult, the (intracranial) jugular foramen is more medially situated, and its course through the bone is more outward than in the infant, so that at its point of exit into the internal jugular vein, its relative position, comparable with that of the infant, is established. The great post-natal development of the mastoid portion of the temporal bone seems to account in great part for this difference in infancy and adult life.

Fig. 1. Medial vertical section through skull and cervical vertebrae of new-born. Parietal bone removed. Lateral sinus laid open. A silver probe, bent slightly downward, enters and passes through jugular vein and bulb. In the adult the passage is tortuous and irregular, so that the probe does not pass.

Owing to the somewhat different course of the bulb through the bone structure, the exposure in very young subjects is never accompanied by the need for disturbing the transverse process of the atlas, since even at its point of greatest depth the outer extremity of this vertebra is still medially situated in relation to the jugular bulb.

The facial nerve overlies the bulb as in the adult. From the point where the Fallopian canal turns downward after passing above the foramen ovale, its course is more backward than in the adult. In the new-born the distance from above the foramen ovale to its exit on the surface of the

Fig. 2. Transverse vertical section through skull of new-born, left side in a plane ¼ inch more anteriorly to right. J. Jugular bulb at its greatest diameter. J.1 Lateral sinus turns forward into jugular vein. L. L. Lateral sinus. T. Transverse process of atlas. M. Medulla and pons. Facial nerve emerges ⅛ inch anterior to plane of this section at F.

skull is only ½ to ⅜ inch. (In the adult it is ¾ inch or more.) In this distance the facial nerve in the new-born follows a course of nearly 45 degrees from the vertical. It emerges from the skull ¼ inch behind, ⅜ inch below and about ½ inch actual distance from the bony tympanic ring.

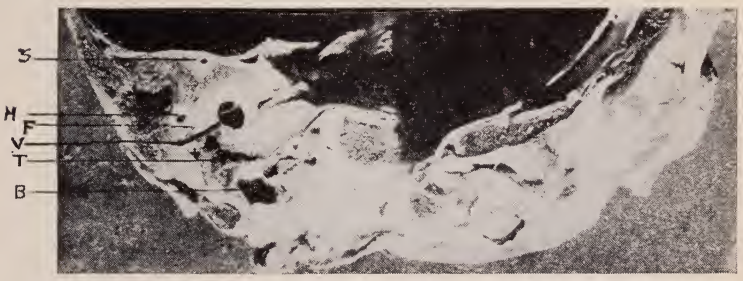
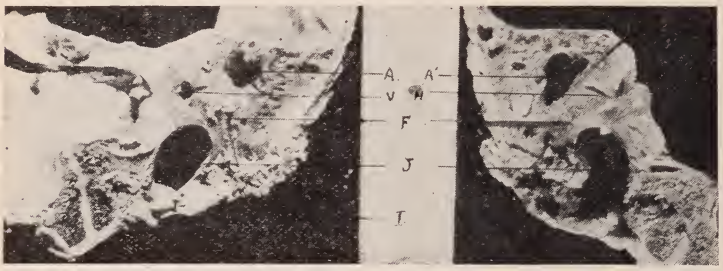


Fig. 3. Transverse vertical section through temporal bone of new-born, same as in preceding figure, but on left side a section has been made in a plane parallel to last, ¼ inch anteriorly. S. Superior semi-circular canal. H. Horizontal semi-circular canal. F. Facial nerve. T. Tympanic cavity. B. Jugular bulb, anterior expanded portion.

In reaching the jugular bulb in the young infant no bony floor of the auditory canal exists to be cut away and the vessel in its course through the bone does not extend so far anteriorly. The bony base of the cranium of the new-born is thinner and the vessel in its forward and downward course emerges rather posteriorly to the tympanic floor. In the infant during the first months, at least, exposure of the sigmoid sinus may be carried downwards to the jugular bulb and its posterior portion may be exposed with less difficulty than in the adult.



Figs. 4 and 5. Transverse vertical section through right temporal and occipital bones, both surfaces of section shown. Fig. 4 shows the smooth dura covering posterior surface of left petrous and basilar portion of occipital bone (in white). The darker portion is that cut by saw passing through wide portion of jugular bulb. J. Opening beneath I into internal jugular vein. F. Facial nerve continuing downward. V. Vestibule and non-ampullary opening of horizontal semi-circular canal. A. Aditus and (Fig. 5). A^1. Mastoid antrum. H. Horizontal semi-circular canal to right of which is seen conjoined horizontal and superior semi-circular canal and below ampullary end of posterior semi-circular canal. In Fig. 5 the lateral sinus enters from behind into the jugular bulb near its lower portion.

The chief differences noted between the sigmoid sinus and jugular bulb of the new-born and the adult are, viz.:

In the infant the sinus is straighter and relatively as well as actually shorter, the jugular vein perforates the bone more directly, with less tortuosity, it is more readily accessible and hence more easily cleared. There is apparently (as noted above) little difference in the size of right and left bulbs in the new-born; though in the five year old child the difference between right and left may be established to as

great an extent as in the adult. At five to six years the styloid process is merely a cartilaginous tubercle. The difference in the direction in the course of the facial nerve in the new-born and adult is due to the rapid post-nasal development of the mastoid portion of the temporal. This, after birth, is rapidly built up around the (nearly) vertical portion of the facial. In other words, the course of the facial nerve as it is found at birth, remains, but the process of development of the mastoid by the superposition of bone to form the mastoid tip and the bony floor of the meatus, lengthens the Fallopian canal in its (nearly) vertical portion only.

The points of difference in the jugular bulb and neighbouring regions of infancy and adolescence worthy the surgeon's notice are:

1. The simple and more direct course and form of the sinus, bulb and vein.
2. The practical equality of size of both bulbs in the new-born.
3. The course of the facial, overlying the bulb as at subsequent years, but passing diagonally **backward** as well as downward through the mastoid.

Discussion.

Dr. B. Alexander Randall, of Philadelphia, called attention to the value of measurement of these parts the separation from the median line—a point which, if he understood correctly, had been omitted from the paper. The tympanic rings at their middle are separated on the average double in the adult what they are in the infant. The average distance of the sigmoid from the posterior wall of the canal, as he pointed out some years ago, is in infants ten millimeters. In the adult because of the curvature of the sigmoid knee dipping deeply into the bone, this is reduced until there may be dehiscence into the canal. Supplementary to Dr. Braislin's valuable investigations, measurements made in the horizontal plane would also have much value.

Dr. Braislin, in closing the discussion, said the point made by Dr. Randall was well taken. The actual as well as the relative distances between the knee and the bulb is surprisingly small in the new born, being only about three-quarters of an inch. After birth this quickly increases because the growth of the head is so enormous in the first five years of life.

A NEW INSTRUMENT TO REMOVE THE NASAL WALL IN CASES OF EMPYEMA OF THE MAXILLARY ANTRUM.

By HENRY A. ALDERTON, M. D., Brooklyn, N. Y.

The usual methods of operating for the evacuation of purulent collections within the antrum of Highmore, through the nasal wall, are; first, by way of the inferior meatus without removal of the inferior turbinated body, either by simple puncture with the trocar and canula or by making a larger opening with various instruments designed for the purpose; secondly, by the removal of the anterior two-thirds of the inferior turbinated body, followed by perforation and removal of a fair-sized section of the underlying nasal wall of the antrum.

Simple opening with the trocar and canula, while suitable for those cases which are acute and under the personal care of the specialist, is inapplicable to cases of long standing or to those cases which come from a distance and in which the after-treatment has to be left in the hands of the general practitioner. Also the washing out of the antral cavity through the small opening, notwithstanding the preparatory application of cocaine solutions, is usually more or less painful from the difficulty in finding and introducing the washing canula; in the hands of the general practitioner this becomes largely a matter of luck. It is well then, when the inferior meatal wall is made the site of attack, to make the opening sufficiently large to abolish this difficulty of after-treatment.

The more complete operation, always desirable in chronic cases, has heretofore been largely done by punching out a hole, after removal by the scissors and snare of the anterior two-thirds of the inferior turbinated body, and then enlarging this small opening by the use of variously designed bone cutting instruments or by the use of a bone rasp. Considerable time is taken in enlarging the original open-

ing and, where the nasal wall curves strongly outward, the procedure is not always so simple as one could desire; especially is this true when there is considerable hemorrhage.

Having in view all these facts, the writer has designed and had constructed an instrument which has given him satisfaction. He used as a model Kerrison's bone punch for mastoid work, simply making a few changes to fit the different requirements: first, the handles were changed in shape so as to fit the hand-grasp better and a spur was placed on the proximal handle against which pressure can be exerted to force the punch blades into and through the bony nasal wall; second, the cutting part was formed by vertically splitting a trocar in half, across from edge to edge, the base of one-half being attached to the distal end of the stationary shank and the base of the other half attached to the distal end of the sliding shank. Both halves were hollow ground, the edges and points sharpened and were held apart by double springs between the handles. The instruments were made in two sizes, the blades being 7 m. m. long in the smaller and 12 m. m. in the larger.

The smaller instrument was designed for removing the nasal wall in the inferior meatus, without destruction of the inferior turbinated body or, in children, with removal of that body; the larger was designed to be used only after the inferior turbinated has been exsected in its anterior two-thirds. Should the nasal septum be deviated toward the affected side, then it is necessary to remove the inferior turbinated before it is possible to use the smaller instrument.

To make the opening, with the smaller instrument, in the inferior meatus without removal of the inferior turbinated body, the parts are anæsthetized and devascularized by cocaine and adrenalin solutions, the instrument is introduced between the septum and the inferior turbinated, with the blades closed and the points down, until the blades reach a point two-thirds of the distance backward of the inferior turbinated body; while the shank is held parallel to the nasal floor in this position the blades are permitted

to separate to the full extent of the action of the spring, whereupon the points are rotated under the edge of the inferior turbinated until the blades are horizontal and they are then forcibly plunged through the nasal wall, which at this point is usually thinnest; after the blades have entered the antrum as far as they will go, they are brought together so that they would just meet but for excised plate of bone between them; the instrument is then withdrawn by rotating the blades back to a vertical position with the points down and removed as on introduction. An opening 6 by 12 min. is thus made in the nasal wall in a very few seconds; amply large enough to avoid the difficulty in after-treatment that attends introduction of the washing canula.

In chronic cases, where a larger opening is desirable, the anterior two-thirds of the inferior turbinated body is removed and then the large-sized punch is introduced into the nasal passage with the points of the closed blades up and the shank close to and parallel with the nasal floor. The blades are passed in to a point opposite the beginning of the posterior third of the inferior turbinated body, opened and rotated toward the nasal wall and, when horizontal, forced through into the antral cavity, brought together just so as to hug the included bone snugly and by a reversal of these motions withdrawn. The blades must not be pressed with the full strength of the hand against each other, as this is not necessary and might result in the fracture of the distal blade in case of any flaw in the material. The opening left measures 6 by 14 min., but is often much larger from the fracturing of the bone around the cutting edges.

One great advantage in this instrument lies in the fact that hemorrhage offers no impediment to its use; the operation can be done just as well by touch as by sight, by marking the extent of introduction of the blades by a piece of plaster on the stationary shank. Partly because of this, the instrument is especially adapted to chronic cases when it is desirable to use general anæsthesia.

The instrument is made by E. B. Meyrowitz of New York City.

Discussion.

Dr. James F. McCaw, of Watertown, N. Y., referring to the statement of Dr. Alderton that he goes into the antrum without removing any part of the inferior turbinate; it would seem that drawing these cutting plates together, would injure the inferior turbinate, as the space between the floor and the lower border of the turbinate would seem insufficient to prevent it.

Dr. Alderton, in closing the discussion, said there is usually sufficient shrinkage of the soft tissue of the inferior turbinate body to allow the passage of this instrument, but suppose one does a little injury to the tissues, there is less injury to mucous membrane than would result from removal of the anterior two-thirds of the turbinate body. The only case in which it was impossible to use it was with large deflection of the nasal septum to the affected side.

THE RELATION OF ACTIVE AUTOINTOXICATIONS TO ACUTE MIDDLE EAR AND MASTOID INFLAMMATIONS.

By Sargent F. Snow, Syracuse, N. Y.

GENTLEMEN:—In the study of a diseased condition we are doubly armed if we have a clear conception of its determining cause.

Knowing the determining cause and **seeing** easily the local manifestation, puts effective weapons in our hands so that we may attack the trouble from two ways.

A double leverage like this is very valuable when we have under treatment acute affections that are liable to develop and extend into vital regions.

Acute Middle Ear and Mastoid Inflammations are well within the list of diseases that call for serious thought, and a suggestion or plan of treatment that will give us increased power over these conditions, I am sure, will be welcomed by all.

For years specialists have studied and discussed the local manifestation of mastoiditis and their calls for relief to the end that we now have splendidly developed and effective plans for local treatment. In fact, mastoid operative technique is so perfected by experienced operators, that but little concern need be felt for their cases, and perhaps it matters little with these operators whether the etiology of the trouble is considered or not,—their patient is quite sure of ultimate recovery from the blood letting and good drainage they obtain.

The question I would put before you now is whether we cannot modify the danger and cut short the duration of the trouble by working at both the cause and effect. In other

words, working from both ends towards the middle; treating the fundamental cause of the inflammation as vigorously as we treat its results.

Previous to 1909 I was content to treat my acute mastoid cases locally, not giving much attention to the constitutional state, except perhaps a perfunctory order for a cathartic at the beginning of treatment. A closer study of late into the causes of inflammation of the head membranes has given me a different view point, and I am happy now in the thought that I am much better equipped for a battle against these inflammations.

The whole question of local congestion seems to revolve around and rest upon the state of our glandular activities. If our antibodies,—nature's army of opsonins,—are in normal quantities and in normal activity, they fight down and keep under subjection bacteria that are always ready somewhere in our economy for pernicious development.

A basic idea in medical philosophy of long ago was that nature provided man with defensive agencies to combat disease.

Sir Almorth Wright's development of the opsonic theory and his research showing that the blood serum contains protective substances that act upon pathogenic bacteria, adds further proof.

Metchnikoff has made plain the importance of the protective body cells against microparasites.

The researches of Ehrlich, Brieger and others have shown us the relations between antigens and antibodies; Hektoen of Chicago,* locates the probable formation of antibodies as in the spleen and other lymphoid structures; all showing that medical science is rapidly unfolding important truths.

The presence of bacteria **per se** is not sufficient to cause acute inflammations;—the soil surrounding them must be favorable to their propagation;—the cellular structures must be so modified in functional activity as to be no longer able to keep the pernicious bacteria under subjection.

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*Transactions of the last Congress of American Physicians and Surgeons, Page 10.

It is well known that various bacteria may be present in the tympanic cavity without exciting inflamation until the conditions of the secretions and tissues are favorable to their rapid propagation. Those conditions favorable to the rapid propagation of dormant but vicious bacteria is, in my opinion, induced by an active autointoxication or crisis of elimination. Membrane congestions, in the Author's opinion, with few exceptions, are practically autotoxic expressions; these antotoxic expressions come with more or less periodic regularity, and any organ or sinus covered or lined with a membrane is in danger.

Closer observations on acute pharyngeal, nasal sinus, middle ear and mastoid inflammations, astound me at the invariable relation evident between those inflammations and an active autotoxic state of the system. Frequently we find that there have been weekly evidences that they were self poisoned; evidences made plain by digestive disturbances or recurring head colds.

Each case may not show all the symptoms of this etiological factor, but the skilled observer will detect some of them. The local signs of autointoxication depend largely upon the individual;—sometimes the complexion is perfect, but the eyes or posterior one third of the tongue will give the evidences; not every one will show a tongue coated to the tip, but he will show some of the other well marked principal symptoms;—by drawing down the lids of the eye his conjunctiva will be found of a yellowish cast, or his skin may be gray or ashen, his breath foul, or his tongue dry and reddened;—all proving that the patient's system, from some cause or other, has accummulated a large load of enterotoxins. This load of poisons may be the result of several months of inactivity, constipation and over-eating;—it may be the result of recent dietary excesses, or from overstrain and exhaustion, causing poor action of the liver and digestive functions;—whatever the source, nature demands relief from the stored up toxins.

Autointoxication has a multiplicity of causes, each one leading up to a time when the system can no longer tolerate

its load of poisonous substances, the different organs and membranes of the body are forced to do, and will do, double eliminative work until some organ fails or flags in its function;—then we have an acute inflammation in some part of the body from the faulty metabolism;—an autotoxic crisis. This is the time when our cellular structures are so weak in their functional activity that germs are able to multiply with impunity.

Should our tonsils, accessory sinuses or tympanic cavities be harboring staphlococi, streptococi, or any variety of bacteria, these vicious trouble producers are apt to soon be multiplying at a rapid rate, opposed by only a few weakened, attenuated antibodies. The term "attenuated antibodies" is chosen advisedly, because for days, perhaps weeks, the blood, secretions and tissues of this patient have been getting so surcharged with poisons absorbed from his own intestinal tract, that his army of fighting antibodies, usually capable, have lost their strength in their efforts against his own autointoxication;—he then becomes a prey to diseases from bacteria originating outside his body,—some fulminating, acute, inflammatory process of varying virulency.

It seems to be largely an accident as to where this inflammation shall be located;—determined, I assume, by the place in the body where vicious bacteria are most numerous, or causes for local congestion are most prominent. Should our eustachian tube be closed by an autotoxic cold, should our nasal accessory sinuses be shut off from ventilation and drainage by a congestion in the nose, we have then just the condition proper for rapid development and suppurative processes with attendant consequences.

When we see a marked autotoxic appearance in our patients, we may expect soon an acute inflammation of some degree in some part of the body.

According to Coombe on Autointoxication, (Page 128,) it seems to depend upon the individual predisposition which organ or part will suffer. He says "In one it will be the nervous system, in another the lungs, in a third the heart,

etc. The gradual accumulation of these poisons in the organism will, at variable intervals, determine crises of elimination, according to the nature of the sufferer; at times a febrile access, at others a cutaneous outbreak, or an intestinal discharge; all of which leaves the patient weakened, but otherwise improved for a certain length of time, until a reaccumulation reproduces the same symptoms at necessarily varying intervals.''

Let no mistake be made from this paper and the author construed as meaning that, as a rule, local surgical measures are contra-indicated. In fact, in acute mastoiditis, surgical measures are indicated in nearly all the cases that get to the ear specialist. It does not follow, though, that the external mastoid operation will have to be performed in all cases showing tenderness and swelling over the mastoid region; experience has shown that a very large percentage of such cases will recover with the drainage obtained by a free incision of the tympanic membrane, attic folds and periostium of the superior canal wall. The kind of operation and the amount of drainage necessary to obtain, will depend upon the exigencies of the case.

Acute middle ear and mastoid inflammations are clearly of a dangerous, active, fulminating order, and the bacteria present are apt to be of such a virulent nature that constitutional measures alone are **not** safe to rely upon.

A point the author would make in this paper is that there are certain alteratives that can cause the blood to become bacteriolytic;—certain medicines that seem to act as antigens and induce the formation of more antibodies, or at least rehabilitate them with more antitoxic power.

Any substance capable of inciting the formation of antibodies is an antigen; mercury, iodine, the adrenal extractives, etc., seem to be in this class and are hopeful adjuncts in this 20th century fight against disease. Vaccine therapy also holds out some hope, but the indications are not very encouraging in acute mastoid troubles. The author relies at present most upon mercury by the mouth in some form as a remedial agent.

Authorities seem to agree that antitoxic elements are produced in the lymphoid structures of the body and also that mercury is our most efficient medicinal stimulant of systemic functions. Mercury then can very properly be called an antigen and clinical experiences of my own would seem to prove that it surely has antigenic properties; properly given, it appears to act as an antigen, stimulating the formation of antibodies to ward off and subdue disease.

Sajon says,* "Certain alteratives can cause the blood to become bacteriolytic and antitoxic by provoking the formation and accumulation therein of more or less antitoxin."

As time develops, it is probable that our knowledge as to how to use these alteratives will improve. At present, I find myself prescribing calomel in 1-10 gr. doses, frequently repeated until a grain or a grain and a half have been given, then after making sure the bowels are sufficiently active, it is followed by a dose of castor oil or salines. This plan is repeated in two days and sometimes the very next day if the first has not been satisfactory, and it is continued every second day until active symptoms are subsiding. It is possible smaller doses more frequently repeated, would be better for antigenic action. During convalescence, either following the internal or external mastoid operation, I prescribe calomel in good dosage at least every five days, not for its cathartic action alone, but for its double action as an intestinal cleanser, glandular stimulant and corrective for the whole system.

Since adopting this more vigorous plan of constitutional treatment and attacking the cause of acute mastoiditis as promptly and thoroughly as the effect, I find that recovery is made in at least one-half the time, and with one-third the effort than could be obtained under my former plan of using, practically only local treatment.

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*See Vol. II, Page 1136, "Internal Secretions."

Discussion.

Dr. Joseph A. Kenefick, of New York City, said Dr. Snow drew a typical picture of a patient with auto-infection, such as the speaker described in discussing Dr. Bryant's paper. The cases which, in his experience, are particularly liable to infection from influenza are those which are suffering from auto-toxic conditions such as Dr. Snow described. His treatment in these cases has been successful in preventing the occurrence of acute middle ear disease and other complications by means of calomel and sodium salicylate. He emphasized the importance of salicylate of soda with calomel. Theoretically no drug should be combined with a metal chloride, but he would not hesitate to combine these two. He had had extraordinary success in aborting ear and sinus troubles, in cases of auto-intoxication, and had succeeded in bringing to prompt conclusion, threatening evidences of severe influenza.

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—OF—

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Dr. Edmund C. Rivers.....1632 Welton St.....Denver, Col.....

Dr. Arthur W. de Roaldes...624 Gravier St.....New Orleans, La.....

Dr. Walter Roberts*.....1732 Spruce St.....Philadelphia, Pa.....

Dr. William K. Rogers....88 East State St.....Columbus, Ohio.....

Dr. D. B. St. John Roosa.

Dr. John D. Rushmore.....129 Montague St.....Brooklyn, N. Y.....

Dr. T. Laurance Saunders*.43 West 50th St.....New York City.....

Dr. Wm. W. Seely.

Dr. Ralph W. Seiss.....255 South 17th St.....Philadelphia, Pa.....

Dr. S. Sexton.

Dr. George E. Shambaugh..People's Gas Bldg.....Chicago, Ill.....

Dr. John B. Shapleigh.....Grand & Washington Aves..St. Louis, Mo.....

Dr. Warren S. Shattuck, Jr.160 Clinton St.....Brooklyn, N. Y.....

Dr. Henry L. Shaw.

Dr. John E. Sheppard.....130 Montague St.....Brooklyn, N. Y.....

Dr. Burt R. Shurly*.....32 Adams Ave. W.....Detroit, Mich.....

Dr. E. Terry Smith.....70 Cove St.....Hartford, Conn.....

Dr. S. MacCuen Smith.....1429 Spruce St.....Philadelphia, Pa.....

Dr. Sargent F. Snow.....707 University Block.....Syracuse, N. Y.....

Dr. Edmund D. Spear.....100 Boylston St.....Boston, Mass.....

Dr. Horatio N. Spencer.....2725 Washington Ave.....St. Louis, Mo.....

Dr. Selden Spencer*.....2725 Washington Ave.....St. Louis, Mo.....

Dr. Frank Sprague.

Dr. Franklin M. Stephens...71 West 49th St.....New York, N. Y.....

Dr. George T. Stevens.....40 East 41st St.....New York, N. Y.....

Dr. Edward Stieren.....Westinghouse Building....Pittsburg, Pa.....

Dr. Samuel B. St. John.

Dr. George Strawbridge....202 South 15th St.....Philadelphia, Pa.....
Dr. Joseph A. Stucky*.....Lexington, Ky.....
Dr. Theron Y. Sutphen....999 Broad St.....Newark, N. J.....
Dr. Lewis H. Taylor.....82 South Franklin St.....Wilkes Barre, Pa.....
Dr. Samuel Theobald.....Cathedral & Howard Sts....Baltimore, Md.....
Dr. William Thomson.
Dr. Robert Tilley.
Dr. J. J. Thomson*.....40 West 47th St.....New York City.....
Dr. Andrew Timberman*...112 East Broad St.....Columbus, Ohio.....
Dr. J. J. B. Vermyne.
Dr. Ernest de Wolfe Wales..423 Board of Trade Bldg.. Indianapolis, Ind.....
Dr. D. Harold Walker.....28 Newbury St.....Boston, Mass.....
Dr. David Webster.....308 Madison Ave.....New York, N. Y.....
Dr. Joseph A. White.....200 East Franklin St.....Richmond, Va.....
Dr. Edward Wheelock.....26 Gibbs St.....Rochester, N. Y.....
Dr. Fred Whiting.....19 West 47th St.....New York, N. Y.....
Dr. Frederick M. Wilson...834 Myrtle Ave.....Bridgeport, Conn.....
Dr. John Gordon Wilson*..104 S. Michigan Ave.....Chicago, Ill.....
Dr. Hiram Woods.....842 Park Ave.....Baltimore, Md.....

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*Associate.

American Otological Society.

The forty-fifth annual meeting of the American Otological Society was held at Atlantic City, New Jersey, at the Hotel Chelsea, on June 10th and 11th, 1912.

The following members were present:

DRS.	A. E. ADAMS	Newburg, N. Y.
	J. L. ADAMS	New York, N. Y.
	JOHN F. BARNHILL	Indianapolis, Ind.
	H. S. BIRKETT	Montreal, Canada.
	J. H. BRYAN	Washington, D. C.
	W. SOHIER BRYANT	New York, N. Y.
	WILLIAM C. BRAISLIN	Brooklyn, N. Y.
	RALPH BUTLER	Philadelphia, Pa.
	CHAS. W. COX	Brooklyn, N. Y.
	J. FRANK CROUCH	Baltimore, Md.
	E. W. DAY	Pittsburg, Pa.
	EDWARD B. DENCH	New York, N. Y.
	ARTHUR B. DUEL	New York, N. Y.
	E. GRUENING	New York, N. Y.
	WELLS P. EAGELTON	Newark, N. J.
	E. E. HOLT	Portland, Me.
	THOMAS HUBBARD	Toledo, O.
	W. H. HASKIN	New York, N. Y.
	DAVID HARROWER	Worcester, Mass.
	FRED L. JACK	Boston, Mass.
	GEO. A. LELAND	Boston, Mass.
	ROBERT LEWIS	New York, N. Y.
	JAS. E. LOGAN	Kansas City, Mo.
	DAVID B. LOVELL	Worcester, Mass.
	JAMES F. McKERNON	New York, N. Y.
	WENDELL C. PHILLIPS	New York, N. Y.
	FRANCIS R. PACKARD	Philadelphia, Pa.
	NORVAL H. PIERCE	Chicago, Ill.
	JOHN B. RAE	New York, N. Y.

RICHARD A. REEVE.....Toronto, Canada.
CHARLES W. RICHARDSON.....Washington, D. C.
RENRY O. REIK.....Baltimore, Md.
W. K. ROGERS.....Columbus, O.
B. ALEX. RANDALL.....Philadelphia, Pa.
J. B. SHAPLEIGH.....St. Louis, Mo.
WARREN S. SHATTUCK, JR....Brooklyn, N. Y.
S. MacCUEN SMITH.....Philadelphia, Pa.
EDWARD STIEREN.....Pittsburg, Pa.
E. TERRY SMITH.....Hartford, Conn.
GEORGE E. SHAMBAUGH.....Chicago, Ill.
D. H. WALKER.....Boston, Mass.
J. A. WHITE.....Richmond, Va.
FRED WHITING.....New York, N. Y.
DAVID WEBSTER.....New York, N. Y.
HIRAM WOODS.....Baltimore, Md.

Associate Members.

DRS. HARRY FRIEDENWALD.....Baltimore, Md.
MAX A. GOLDSTEIN.....St. Louis, Mo.
THOS. HUBBARD.....Toledo, O.
HANAU W. LOEB.....St. Louis, Mo.
P. D KERRISON.....New York, N. Y.
JAMES F. McCAW.....Watertown, N. Y.
SELDEN SPENCER.....St. Louis, Mo.
J. A. STUCKY.....Lexington, Ky.

Guests.

DRS. THOS. J. GALLAGHER.....Denver, Colo.
E. R. LEWIS.....Dubuque, Iowa.
JOHN WESLEY LONG.....Greensboro, N. C.
CULLEN F. WELTY.....San Francisco, Cal

Executive Session.

Monday Morning, June 10th, 10 A. M.

The meeting was declared open by the President, Dr. E. B. Dench.

The minutes of the 1911 meeting were read and approved.

The President appointed Doctors Norval H. Pierce, Chicago, Ill.; James E. Logan, Kansas City, Mo.; S. MacCuen Smith, Philadelphia, Pa., to serve as the Business Committee.

The report of the Treasurer was read and on motion approved.

The following candidates were nominated for membership: Dr. George H. Powers, Jr., Boston, Mass—Proposed by Dr. Frederick L. Jack; seconded by Dr. George A. Leland. Dr. George L. Toby, Boston, Mass.—Proposed by Dr. Frederick L. Jack; seconded by Dr. George A. Leland. Dr. E. M. Holmes, Boston, Mass.—Proposed by Dr. George A. Leland; seconded by Dr. Frederick L. Jack. Dr. C. R. C. Borden, Boston, Mass.—Proposed by Dr. George A. Leland; seconded by Dr. Frederick L. Jack. Dr. R. Bishop Canfield, Chicago, Ill.—Proposed by Dr. George E. Shambaugh; seconded by Dr. B. Alexander Randall. Dr. R. Johnson Held, New York City—Proposed by Dr. Wendell C. Phillips; seconded by Dr. Edward Stieren. Dr. Samuel J. Kopetzky, New York City—Proposed by Dr. Wendell C. Phillips; seconded by Dr. B. Alexander Randall. Dr. John R. Page, New York City—Proposed by Dr. Arthur B. Duel; seconded by Dr. Emil Gruening. Dr. Thomas J. Gallagher, Denver, Colo.—Proposed by Dr. James E. Logan; seconded by Dr. Norval H. Pierce.

The Secretary presented the resignations of Dr. William Lincoln of Cleveland, Ohio, and of Dr. John Van Duyn of Syracuse, New York. On motion the resignations were accepted with regret.

The Secretary reported the death of Dr. P. Davidson of Richmond, Va., and Dr. B. E. Fryer of Kansas City, Mo.

Appropriate remarks were made by the President.

The Secretary reported that the name of Dr. Wheelock Rider, Rochester, New York, had been changed to Edward Wheelock.

The Committee on "The Deaf Child" submitted a supplementary report. On motion the report was accepted and the Committee discharged with thanks.

The Business Committee reported, and after adjournment to Scientific Session, papers were read by the following members: Dr. Bryan, Dr. Randall, Dr. Rae, Dr. Pierce, Dr. Shambaugh, Dr. Bryant, Dr. Gruening, Dr. Richardson, Dr. Lewis, Dr. Jack, Dr. Reik, Dr. Barnhill, Dr. Eagelton, Dr. Whiting, Dr. McCaw, Dr. Goldstein, Dr. Theobald, Dr.

Bryant, Dr. MacCuen Smith, Dr. Hubbard, Dr. Holt, Dr. Stucky, Dr. Braislin, Dr. Packard.

Monday Afternoon, June 10th.

Excuses for absence were submitted by the following members: Dr. H. P. Mosher, Dr. Blake, Dr. Berens, Dr. Thos. J. Harris, Dr. Gorham Bacon, Dr. Geo. L. Richards, Dr. J. C. Lester, Dr. Geo. McAuliffe, Dr. C. G. Coakley, Dr. Henry A. Alderton.

Dr. Chas. W. Richardson, Washington, was appointed delegate to the Congress at Washington—1913. Dr. Joseph H. Bryan, Washington, was appointed alternate.

The Scientific Session was then continued.

Tuesday Morning, June 11th.

The report of the Membership Committee was received.

On motion the following gentlemen were elected to membership in the Society: Dr. J. J. Thomson, New York City, N. Y.; Dr. Hugh B. Blackwell, New York City; Dr. T. Lawrence Saunders, New York City; Dr. O. A. M. McKimmie, Washington, D. C.; Dr. Alfred M. Amador, Boston, Mass.; Dr. G. Hudson Makuen, Philadelphia, Pa.; Frank T. Hopkins, New York City; Dr. Stephen H. Lutz, Brooklyn, N. Y.; Dr. John Gordon Wilson, Chicago, Ill.; Dr. B. L. Shurly, Detroit, Mich.

On motion the following were carried over for one year: Dr. Samuel McCullagh, New York City; Dr. Antonie P. Voislawsky, New York City; Dr. Chas. P. Perkins, New York City.

The Business Committee acting on nominations submitted the following report:

President—James F. McKernon, New York City.

Vice-President—Eugene A. Crockett, Boston, Mass.

Secretary-Treasurer—John B. Rae, New York City.

Membership Committee—Edward B. Dench, New York City; Ewing W. Day, Pittsburg, Pa.; George E. Shambaugh, Chicago, Ill.

Publication Committee—B. Alexander Randall, Philadelphia, Pa.; Arthur B. Duel, New York City.

On motion the Secretary cast one ballot for these gentlemen and they were accordingly declared elected.

The Secretary read a letter from the "Society on Internationalism."

After discussion it was moved and carried that the matter be referred to the President and Secretary with power to act.

A letter was read by the Secretary with reference to Standardizing Disinfectants. On motion, duly seconded and carried, the letter was laid on the table.

The President appointed as the Program Committee for next year, Dr. James F. McKernon, New York City; Eugene A. Crockett, Boston, Mass.; Robert Lewis, New York City.

The Scientific Session was then continued, at the close of which the Society adjourned.

ON THE RELATION OF DISEASES OF THE POSTERIOR SINUSES TO PAINFUL CONDITIONS OF THE EAR.

By J. H. BRYAN, M. D., Washington, D. C.

Earache, resulting from some irritation in the course of the dental nerve, such as dentition in the infant, or caries, or inflamed nerves, in those who have passed the eruptive period of teething, has been known for a long time, but pain in the ear as a result of reflex irritation in consequence of some diseased condition of the posterior sinuses, is a subject of which I find no mention made, either in the literature on diseases of the sinuses, or in that on diseases of the ear. The only author whose writings I am familiar with, who has alluded to this subject at all is St. Clair Thompson, and he mentions severe earache as a prominent symptom in a report of one of his cases of abscess of the sphenoid sinus. It seems strange that after so much work has been done in recent years on diseases of the accessory sinuses, that no mention of this condition has been made. If other observers have met these painful conditions of the ear resulting from sinus disease they have either not taken the trouble to record their observations, or have done so in such an obscure way as not to attract attention.

Referring to the anatomy of the nose and naso-pharynx shows this region very rich in sympathetic nerves. We find two important ganglia or stations which serve to receive and transmit impressions, not only to the forward part of the nose, but also to the pharynx and the ear. The spheno-palatine ganglion, also known as Meckels, is found in the upper part of the spheno-maxillary fossa, lying just under the sphenoid sinus. This ganglion has for one of its main branches the vidian which is made by the union of the great superficial petrosal and the great deep petrosal nerves. As the vidian passes through the canal of the same name it gives off one or more branches to the otic ganglion, which

lies on a lower level and on a plane posterior to the sphenoidal sinus. This is Arnold's ganglion, and is situated just below the foramen ovale. This ganglion throws off several branches to the mouth of the eustachian tube, and also one or two filaments which inosculate with the auriculo-temporal nerve, and a communicating branch to the cord a-tympani. With such an intricate, and at the same time intimate relation between these two sympathetic ganglia, it is easily understood how irritations resulting from pathologic conditions located in the deeper part of the head, such as in the neighborhood of the sphenoidal sinus and the post ethmoidal cells, can have painful impressions carried to some distant part, such as the middle ear, with which they have no direct relation. Cases in which earache is a symptom of posterior sinus diseases are undoubtedly rare, but they, according to my experience, occur sufficiently often to make a record of them desirable. The pain is simply reflex in character, and does not depend upon any pathologic condition present in the ear. In the several cases that have come under my observation, I have found nothing more than a very moderate form of chronic catarrhal inflammation, and even that has only been present in two of my cases.

Case I. Chronic suppurating frontal, ethmoidal, maxillary and sphenoidal sinusitis, complaining of earache. This patient, a woman of middle age, consulted me, complaining of severe pain in the right ear, which she stated had been going on for several months with varying intensity. She complained of morning nausea and vertigo, but no tinnitus. H. D. W. R. 40/40. Examination showed right membrana tympani normal in color and with normal light reflex. Examination of the nose showed right nasal chamber containing a large quantity of pus in the middle meatus, passing down into the post nasal space, the middle turbinates were also enlarged. The probe revealed caries in the anterior and middle ethmoidal region. Pressure over the supra and infra-orbital ridges elicited severe pain. The electric-light test showed the right side dark, while those on the left side were translucent. Farther questioning elicited the fact

that for a number of years she had severe frontal headaches, and for eight or ten years she had been conscious of a catarrhal condition of the nose, but having failed to get relief she had ceased to complain of them. The earache, however, which appeared recently, was causing her great distress. She was informed that the ear was in a normal condition, and that the earache was due to the trouble in the sinuses, and that her condition was a serious one and that an operation was imperative. Several days before resorting to the major operation, the middle turbinate was removed under local anaethesia, and in doing so a large part of the middle and posterior cells were removed, exposing the sphenoidal sinus, which was found to be filled with pus. The improved drainage resulting from this operation afforded her great relief, and with almost complete cessation of pain in the ear. Within a few days, however, the pain in the head returned with such intensity that she readily submitted to the external operation. The frontal sinus was opened and nearly the whole of the anterior wall removed. The cavity was filled with pus and granulation tissue. After cleaning out the cavity, it was found to be the seat of extensive caries, and a large part of the posterior wall of the sinus had to be removed, exposing the dura to a considerable extent. The ethmoidal cells and the anterior wall of the right sphenoidal sinus were removed and the parts freely curetted. The frontal sinus and the ethmoidal and sphenoidal regions were packed with iodoform gauze, and the external wound closed. This patient made a slow but uninterrupted recovery, with the complete healing of the parts, the reflex pain in the ear subsided, and she has not referred to the pain in the ear since.

Case II. Chronic suppurating frontal, ethmoidal, sphenoidal sinusitis, on the right side, accompanied by severe pain in the corresponding ear before and after the operation. This case, a well nourished man of about forty years, consulted me for severe headache referred to the right frontal region, and also pain in the corresponding ear. Examination showed right side of nose filled with purulent secretion, and marked

swelling of the middle turbinate, nearly closing this side of the nose. The probe revealed extensive caries of the anterior and middle ethmoidal cells on the right side. The electric-light test showed all the cavities on the right side dark, while those on the corresponding side were translucent. Irrigation of right antrum brought away a large quantity of pus resulting from a drainage from the cavities above. Examination of the ears showed nothing abnormal except an undue retraction of the right membrana tympani. Hearing distance normal in both sides. The patient stated that the pain in the ear was of recent origin but had been growing more severe of late. As a preliminary to the external operation the right middle turbinate was removed under cocaine anaesthesia, and after its removal there was found to exist extensive disease of the posterior ethmoidal cells and the sphenoidal sinus. The radical operation was then resorted to, the whole of the anterior wall of the frontal sinus was removed, as well as a part of its floor, and the whole of the ethmoidal cells as far back as the sphenoid. This cavity was then opened and the anterior wall and part of its floor removed. The frontal and the sphenoidal sinuses were found to be filled with thick fetid pus, and the whole of the ethmoid cells were in a necrotic state. After thoroughly cleansing the parts, the cavities were packed with iodoform gauze and the external wound closed in the usual manner. For several days the patient complained of intense pain in the ear, examination of which showed nothing abnormal. Hot applications to the ear and moderate doses of morphia were required for several days when the pain suddenly ceased. This patient made an uninterrupted and quick recovery, being entirely relieved of the sinus disease and all painful symptoms referred to the ear.

Case III. Chronic suppurating pan-sinusitis followed by membranous closure of the left sphenoidal sinus and accompanied by painful sensation in the left ear when the sinus becomes air-bound. It would be tedious to go into the details of this long and troublesome case, and I will refer only to that part which relates to the sphenoidal disease and the

subsequent earaches. The sphenoidal cavities in this case were irregular and abnormally placed. On the left side, and the one that has been followed by ear sequelae, was a fairly well developed sinus located in the upper part of the body of the sphenoidal bone. This cavity was opened by the endo-nasal route and found to be badly diseased. The whole of the anterior wall was removed by means of a cutting forceps. The relief from the symptoms due to the sphenoidal disease was very great, but in the course of time a membranous closure of the cavity took place, a condition which occasionally follows this operation, and from time to time there is an almost complete closure of the cavity. In spite of the numerous attempts to produce a large permanent opening in the sinus the membrane is always reproduced, and when the closure becomes so marked as to produce an air-bound cavity there is nearly always a reflex pain in the corresponding ear resulting. The sinus itself has remained free from disease since the operation except when she has had an attack of influenza when it becomes inflamed, but this condition readily passes off under treatment. There is a moderate degree of chronic catarrhal inflammation of the left middle ear, the membrana tympani showing some retraction, thickening and loss of lustre. The hearing distance is about ten inches below normal. When she has these periodical attacks of earache, enlarging the opening in the sinus and inflating the ear with the eustachian catheter gives almost instant relief. On the right side the condition has been entirely different, there has been no pain referred to the ear, but she has almost constant trigeminal neuralgia, the pain being referred principally to the supra and infra-orbital region. more especially to the infra-orbital ridge near the internal angle. All efforts to reach this sinus by the endo-nasal route failed, as well as the several attempts to reach it through the maxillary route. About eighteen months ago, certain eye symptoms developed which necessitated a radical operation being done. At this operation the whole of the ethmoidal cells, including the inner wall of the orbit, were removed as far back as the sphenoid bone. A moderate sized sphenoidal sinus was found in the most de-

pendent part of the body of the bone and dipping down into the pterygoid process. This is the only case that I have met with clinically that corresponds with an anatomical preparation which I showed some years ago at a meeting of the American Laryngological Association, in which the sphenoid sinus extended some distance into the pterygoid process. The eye symptoms were completely relieved by this operation, but the neuralgic symptoms continued, and from time to time are most distressing. A rather complete history from the sphenoidal side of this case is given in order to show how on one side there may be marked reflex pain in the ear, while on the other side the pain is not referred to the ear at all, but follows the course of the trigeminal nerve.

My purpose in bringing this subject before you is to demonstrate a condition which I believe has been overlooked, or not sufficient consideration given to it. I have seen several other cases of persistent earache, in cases occurring in the practice of other men in which the sinus side of the question had been overlooked, and as soon as the disease in the posterior sinus had been relieved the painful condition of the ear subsided. Trigeminal neuralgia resulting from sphenoidal disease has been recognized for some time, and Sluder in several valuable communications has lucidly demonstrated this, but the reflex pain in the ears, due to the irritation of the vidian nerve, which courses along the inferior border of an ordinarily well developed sphenoid sinus, is a subject which we, as aurists and rhinologists, have not given enough attention.

Discussion.

Dr. Charles W. Richardson, of Washington, D. C., said the paper brings forward an interesting point with reference to neuralgic pain in the ear from sphenoidal disease. It was well to understand the possibility of such symptoms arising in the manner described, and to be on guard for them.

Dr. Sohier Bryant, of New York City, said that reflex pain in the ear may arise from any irritation from the trachea up

to the sphenoid and forward to the tip of the nose and teeth. The syndrome of otalgia involving the geniculate ganglion, described by Hunt, he considered very rare.

Dr. B. Alexander Randall, of Philadelphia, had met with fleeting pain, but had found none of sufficient severity and persistence to require operation.

Dr. Wendell C. Phillips, of New York City, thought reflex pain in acute otalgia quite frequent, but he had found only a few instances of it in chronic cases.

Dr. Hanau W. Loeb, of St. Louis, in a series of specimens studied by him, has found the sphenoid sinus in several instances extending almost to the gasserian ganglion which might account for the pain in the ear.

FURTHER STUDIES AS TO THE AXIS OF THE EXTERNAL AUDITORY CANAL, BASED ON 500 CRANIA.

By B. ALEX. RANDALL, M. D., Philadelphia, Pa.

The descriptive anatomy of the temporal bone as usually published, is based upon specimens chosen as ideal, often from too limited a number; and the text-books vary notably, therefore, in their presentation of the subject. If our views are to be unified and the contradictions explained away, we must construct a sort of composite picture of at least one thousand temporal bones to serve as the basis for our presentation of its average topography and structure; and it is better that these should all be from the adult, since even they will offer all too many variations, then the changes due to age can thus be made all the more distinct and certain. The topography of the rest of the skull can then be co-related and every possible aid secured for the more definite surgical anatomy of the otological field. We must know the averages, but also the maxima and minima, of the operation-room, of the axial direction of the auditory canal, of the course and position of the Facial canal as determinable from the exterior landmarks; and so present the facts that the operator may act neither too gropingly nor too rashly. The symmetries and divergencies of the two temporals of the same individual must also be defined, as Arthur Cheatle promises to do for us at the coming International Otological Congress, if we are to have all the needed help in our difficult field.

The writer has studied these questions for some twenty years past and from time to time presented to this Society the tedious details thus gained, not expecting them to be other than very dry food for anyone, yet believing that they may well be digested by those who can interest themselves in such matters and ultimately by their aid be elaborated into valuable contributions to our anatomical knowledge. The relation of the topography of the tempo-

ral to the general configuration of the skull, which has been studied abroad in a few hundred crania by Bezold, Koerner, Schueltske, Regnault and others, I have therefore determined in 500 crania; the course and relations of the Facial canal in a like number; and now I desire to enlarge in like measure my earlier study of one hundred skulls as to the direction of the canal-axis. This has ceased to have one part of the interest which I ascribed to it eighteen years ago, when operative work about the stapes seemed likely to be important; but it cannot cease to be noteworthy so long as chiselling away the back wall of that canal continues to be an essential and delicate part of the evisceration of the tympanum and mastoid; and the growing surgery of the labyrinth needs improved anatomical basis. If I have proved, as I believe I have, that the descending portion of the Facial nerve is almost invariably vertical in its course from the bend just back of the oval window, it must be usually important to know the relation of the apparent axis of the meatus to this vertical or to the transverse axis of the skull which is at right angles to it. The phrase "apparent axis" of the external meatus is used in this connection because it is not always possible to determine upon the macerated skull, still less in clinical cases, what is the precise axis of the whole tortuous canal; but we can easily decide what is the direction of its more visible and nearly rectilinear outer two-thirds. This I have measured with considerable care by means of an instrument designed for the purpose and reported the findings in a first hundred crania to our meeting in 1894; but it is only of late that I could continue the study and apply the test to the other 400, that we may have these data for a full thousand temporal bones, preferably those as to which all possible other relations had also been determined. The individual results will be elsewhere published; just as I cited them for only the first 122 skulls in my preliminary paper before this Society in 1892, but gave the full findings in a paper before the Pan-American Medical Congress of 1893.

The need of increasing the number of such measurements if the results are to have general bearing, is shown by the

notable difference in some of the figures obtained in the new series as contrasted with those for the first hundred, generally European crania in the Hyrtl Collection of the College of Physicians of Philadelphia. Instead of averaging 10° upward and 10° forward as it passes inward, in the additional 400 skulls the axis of the canal averages less than 5° upward and 6° forward. Hence for the whole series of 500 crania or 1000 temporal bones the upward inclination averages 6.12° on the right and 5.56° on the left; while the forward inclination is 5.45° right and 5.36° left. The presumptive less upward inclination in the "slab-sided" dolichocephalic skull of negroid type is hardly borne out, since the canal is only some 2° more nearly transverse in the long-headed.

The maxima and minima figures remain much the same as in the previous study, but the upward inclination was sometimes wholly absent, even in markedly brachycephalic skulls; and maximum findings up to 20° were met even in extreme dolichocephalic specimens. Extreme results are more common in crania otherwise deformed by disease or racial customs; for I intentionally included some hydrocephalic skulls as well as those of "flat-head" Peruvian or North American Indians. Obliquity of the axis in one plane had little concurrence with such in the other—minimal forward inclination being associated with a maximum in the vertical or vice versa almost as often as not. Symmetry was almost invariable; and when rare discrepancies of 3°—5° were met, they were almost invariably in skulls showing in other respects much more notable asymmetries. Age may be said to have some slight relation in so far as the undeveloped bony canal of childhood naturally lacks the deviation from the transverse axis of the skull which is largely in the outer two-thirds of the adult meatus; but the majority of the crania of aged persons also showed small angles of obliquity. A measurement of about 10° in one or both planes was found in about 100 skulls; but was not proportionately so frequent as in the first series.

This anatomical study substantiates, therefore, the clinical observation that the external auditory meatus, in its bony as well as its soft-tissue portions, generally inclines upward and forward as it passes inward, but may vary from a maximum of 20° in either direction (rarely in both simultaneously) to an exactly transverse or even very slightly backward course. It is fairly symmetrical on the two sides—the discrepancies being less than in most of the other features of the temporal bones. While it has no clear relation to the general topography of the cranium, it is not so nearly a constant as the vertical direction of the descending portion of the facial canal, and has no definite relation to this; yet clinically the surgeon may estimate its direction in comparison with the transverse axis of the head as indicated by the brows, and utilize it in avoiding the facial nerve in operating.

Its relation to the curve of the facial nerve back of the oval window, as marked by the visability through the external meatus of this opening or of the stapes occupyiny it, was again studied in this larger series. In 30% of the thousand temporals it was hidden—even the more visible stapes projecting from it being hardly discernible—while in 15% more it was so nearly hidden as probably to be unseen in the living. In 11% the oval window was fully visible, while in 4% more it was less surely open to view; in 40% it was partly seen so that it could probably have been clinically recognized and utilized as a landmark. In a few cases the presence of the ossicular chain or of part of the drumhead made a positive determination impossible. A considerable upward inclination of the canal generally facilitated the discerning of the oval window—much forward inclination hampered this.

Just as the downward course of the facial canal seemed wholly uninfluenced by that most variable factor in the configuration of the temporal bone, the size of the jugular foramen and its sulcus, so this exercised no recognizable effect upon the direction of the external meatus. It was larger on the right in 256 skulls, doubtfully so in 23 more; was larger on the left in 139, doubtfully in 14 more; and was

equal on the two sides in 69—figures differing little from my findings in the former study, which dealt with some 300 of these same crania—200 additional specimens in the University of Pennsylvania Museums being now substituted for those then measured in the Army Medical Museum in Washington and the Academy of Natural Science of Philadelphia.

Discussion.

Dr. George A. Leland, of Boston, referred to specimens presented by Mr. Arthur Cheatle in New York, in 1908, and said that at least one specimen showed Mr. Hugh Jones' line.

This was an imaginary line "drawn from the outermost part of the exposed external semi-circular canal to the highest point of the floor of the external meatus,"* the facial nerve being always inside of it.

It will be remembered also that several years before, the late Dr. Charles J. Kipp showed before this society a specimen of a temporal bone, in which the facial nerve passed outward and downward instead of vertically downward, and was in a position where it would most certainly have been wounded without the greatest of care, and was in such an anomalous position that in operating most men would have hit it, confidently expecting it to be in its usual position.

Dr. Randall, in closing the discussion, said there was probably no hard-and-fast rule with reference to the temporal bone in any part. He believed the course of the facial nerve to be one of the most constant features, its vertically descending course being as nearly dependable as any other point. His work had been almost entirely upon the intact skull, because it was impossible to determine the course of the facial nerve otherwise. If the facial nerve ran outward twenty degrees or more it might still be more vertical than Schwartze suggested. The speaker had been unable to prove a variation of more than fifteen degrees. The specimen of Berens, with

* Some points in the Surgical Anatomy of the Temporal Bone Bearing on the Spread and Cure of Suppuration, by Arthur Cheatle, F. R. C. S., London, Archives of Otology, Vol. XXXVII, (No. 5) p.343

apparent anomaly of position of the facial nerve, had not satisfied him that it was a real deviation, despite careful study.

Dr. John F. Barnhill, of Indianapolis, said he had had the Berens specimen in his possession for months and did not believe there was any opening at the stylo-mastoid foramen, but that the nerve made its exit from the bone where one would ordinarily penetrate in doing the mastoid operation. He believed the perpendicular position of the nerve to be very constant but thought there might be anomalies.

THE COURSE OF ACUTE DIFFUSE SUPPURATION OF THE LABYRINTH.

By JOHN B. RAE, M. B., C. M. New York, N. Y.

Suppuration of the labyrinth is acute or chronic, diffuse or circumscribed and occurs in the course of acute or chronic otitis media. The acute circumscribed form is more liable to be a complication of a chronic suppurative otitis media, especially if a feature of that disease be the formation of cholesteatoma. The symptoms of acute invasion of part of the labyrinth last only a few days, are quickly forgotten by the patient and in course of time the *acute* circumscribed becomes a *chronic* circumscribed and the evidences of its presence are to be discovered only on experimental examination. The chronic *circumscribed* may become *diffuse* at any period of the disease by extension to the remaining parts of the labyrinth.

Acute diffuse invasion of the labyrinth occurs in either acute or chronic otitis media, but the most marked type is that occurring during an acute otitis in which the disease progresses with great rapidity and death from diffuse leptomeningitis occurs within a few days. Many of these so-called fulminating cases were formerly reported while the pathology was still obscure, and it is not a matter of surprise that the route of infection through the labyrinth was overlooked in a certain proportion of them. It is now quite certain that in every such case a careful investigation of the labyrinth will be made.

The undiscovered or unoperated acute diffuse labyrinthitis does not necessarily terminate fatally. Symptoms may rapidly subside and the patient be able to resume his daily affairs, conscious only, perhaps, that in addition to the persisting discharge from his ear he has lost the remnant of hearing which he had retained prior to the attack.

A careful functional investigation should be made of all cases in which the radical operation has been performed and

which apply for treatment on account of continued discharge from the operated ear. A proportion of these cases will show complete destruction of the labyrinth, especially if the operation has been performed several years ago. Many cases have been submitted in the past to radical operation without investigation of the hearing, and it is only within very recent years that any routine investigation of the vestibular apparatus has been attempted. It is now a well recognized rule that every chronic discharging case before being submitted to radical operation shall undergo a searching functional examination of both the hearing and the vestibular apparatus. This will differentiate the cases with the lesion confined to the conducting mechanism from those in which, in addition to that, the labyrinth has undergone invasion, and will enable the operator to decide in which cases the radical operation is called for, and in which cases the labyrinth operation is indicated. Modern otology teaches that the chances of the patient suffering from suppurative middle ear with labyrinthitis are better if left alone than if the radical operation is performed without removing the diseased labyrinth.

The observations to be made in the course of a complete acute invasion of the labyrinth will have reference (1) to the general condition of the patient, (2) to the local ear condition as discovered by physical examination, (3) to the spontaneous evidences of labyrinthine involvement, (4) to the functional examination of the hearing and (5) to the experimental investigation of the vestibular apparatus.

(1) *General Condition.* This will depend not so much on the immediate labyrinthine invasion as on the precedent conditions. Two points, however, seem to be of special importance; the presence of temperature above 100° and the complaint of the patient of headache. Temperature is not significant of labyrinthitis and when present and associated with headache, of which the patient complains bitterly, the most careful scrutiny is demanded for any evidence of the onset of meningitis. This headache is not earache, nor is it the pain in the temporal region when the mastoid is involved,

nor will it ever be described by the patient in a way to lead to confusion with either.

Some difficulty may arise with regard to the value of the temperature when the labyrinthitis complicates an acute middle ear in which we naturally expect some temperature. Observation after free myringotomy will reveal the true significance.

(2) *Local Ear Conditions.* Nothing need be said with regard to these. The examiner will at once determine whether he has to deal with an acute ear or a chronic ear and will observe when to be found the presence of granulation, poplys or cholesteatomatous material. The special significance of a marginal perforation in the upper posterior part of the drum is well known.

(3) *Spontaneous Evidences of Labyrinthine Involvement.* These refer wholly to the vestibular apparatus and are to be observed only over a very few days. They include (a) vertigo, with nausea and vomiting, (b) spontaneous rotatory nystagmus and (c) disturbances of equilibrium.

They are all due in the first place to irritation of the end organ of the vestibular nerve on the deceased side. The patient is dizzy even when lying in bed with the eyes closed. The vertigo is accompanied at the onset by nausea and vomiting. The spontaneous rotatory nystagmus is to the diseased side, following the law that stimulation of a centre on one side gives rise to nystagmus to the same side. This nystagmus can at first be observed in any position of the eye. It will, of course, be increased in intensity on abduction in the direction of the quick component. Nausea and vomiting quickly cease and the spontaneous nystagmus diminishes in intensity so that after 24 or 48 hours—depending on the rapidity of extension of the disease and the destruction of the end organs—it may have entirely disappeared or be evident only on extreme abduction.

The disturbances of equilibrium follow the usual law of such when of vestibular origin and associated with nystagmus. The patient tends to fall in the direction opposite to

the quick component of the nystagmus and the direction of falling can be altered by changing the position of the patient's head.

As the disease progresses the delicate terminals of the vestibular nerve are destroyed and cease to convey stimuli to the centres. Nystagmus to the side of the bad ear which has been gradually diminishing in intensity, must necessarily cease entirely with the complete destruction, or paralysis, of the end organ. There now results an imbalance of the tonus of the centres on the two sides with the temporary result that the activity of the centre on the good side is in excess of that of the other. There accordingly now is brought about a spontaneous rotatory nystagmus to the *good* side. At first of great intensity this nystagmus is to be observed in all positions of the eye. Within 24 to 48 hours, as equilibrium of the centres is gradually restored, this spontaneous nystagmus diminishes in amount and as it does so, will be best observed on abduction of the eye in the direction of the quick component.

This nystagmus to the good side may also be accompanied by some dizziness and even with vomiting, but neither will be so marked as on the invasion of the labyrinth when the spontaneous symptoms could be directly attributed to the diseased ear.

(4) *The Functional Examination of the Hearing.* The importance of the hearing examination cannot be exaggerated. By means of it in conjunction with the vestibular examination we determine whether or not the labyrinthis is diffuse. Remembering the rapidity with which the acute disease progresses the tests must be repeated at very short intervals. In the earliest stages while the spontaneous nystagmus is still to the diseased side, the funtional examination may give us the evidence of a lesion in the conducting mechanism. A few hours later, Corti's organ meantime having undergone destruction, there may be absolute loss of hearing.

With the good ear excluded by some suitable apparatus, the bad ear should be tested for the voice. The hearing for both low and high notes should be investigated, the abso-

lute bone conduction determined and the Weber and Rinne tests applied. In the earliest stages the functional examination may be just what we would expect from the middle ear condition. The voice may be fairly well heard, the patient be deaf for the low notes, hear the high notes well, have increased bone conduction, have a negative Rinne and refer the Weber to the bad ear. With complete destruction of Corti's organ the picture will be entirely different. There will be complete loss of hearing for the voice. No fork will be heard either by air or by bone conduction and the Weber will be referred to the good ear. The importance of repeated examinations at short intervals is again emphasized.

(5) *The Experimental Investigation of the Vestibular Apparatus.* The different vestibular tests cannot readily be applied in the earliest stages of an acute diffuse labyrinthitis while the patient is extremely dizzy, nor are they of very much importance. While the spontaneous nystagmus is to the bad side it is very evident that the vestibular apparatus on that side is still functionating and we are in need of no experimental testimony in proof of it. When however this stage has passed and the spontaneous nystagmus is to the good side it is also a reasonable conclusion that the vestibular apparatus on the bad side is no longer functionating. In support of this we may endeavor by the caloric tests to influence the spontaneous rotatory nystagmus.

Thus let us suppose that a patient with a suppurating left ear has been under observation, showing evidence of an acute invasion of the left labyrinth. The spontaneous rotatory nystagmus to the left has ceased and the patient is now exhibiting a spontaneous rotatory nystagmus to the right. How can we by the caloric experiments influence this nystagmus? If we irrigate the left ear with hot water and the left apparatus be functionating we may be able to diminish the spontaneous nystagmus to the right to an appreciable extent. Should the apparatus be destroyed there can be no change in the spontaneous rotatory nystagmus to the right.

If we irrigate the left ear with cold water and the apparatus be functionating we may appreciably increase the spontaneous

rotatory nystagmus to the right. Should the apparatus not be functionating there will be no alteration in degree of the spontaneous rotatory nystagmus.

These tests at best under such circumstances are very indefinite and the writer maintains that they are not essential to an understanding of the condition of the vestibular apparatus in the acute form of labyrinthine invasion. It seems a fair conclusion if we reason that while the spontaneous nystagmus is to the diseased side, that apparatus is still functionating, and when in the course of a few hours the spontaneous nystagmus changes to the good side, that the vestibular apparatus on the diseased side has ceased to functionate.

The presence of a fistula is not a common occurrence in acute labyrinthitis but compression and aspiration may be readily practiced to find whether or not a fistula is present.

The diagnosis of acute diffuse labyrinthitis in uncomplicated cases is not difficult and is made when by functional examination and by observation of spontaneous symptoms it is evident that the end organs of both the cochlear and vestibular nerves have ceased to functionate. Thus in the course of an acute or chronic otitis media the diagnosis is made when, associated with spontaneous rotatory nystagmus to the good side, we find absolute loss of hearing on the affected side. Should time have elapsed sufficient to restore the balance of the vestibular centres and no spontaneous nystagmus be present the diagnosis is equally easily made when the functional examination reveals complete loss of hearing on the affected side and when the vestibular apparatus is found incapable of stimulation by the caloric tests.

In each of these cases we are of course presuming that there has immediately preceded a definite train of symptoms, vertigo with nausea and vomiting, disturbances of equilibrium and spontaneous nystagmus.

The most frequent complications which are directly traceable to labyrinthine suppuration are meningitis and cerebellar abscess. Of these, meningitis is immediate and urgent and almost invariably fatal, and it is with this complication

in mind that the question of operation must be decided. With a very limited personal experience but with some knowledge of the work of his colleagues, the writer ventures to take the position that every case of acute diffuse suppuration of the labyrinth should be submitted to operation as soon as the diagnosis is made. Further, with temperature above 100°, and with persistent headache, with or without other evidence of the onset of meningitis operation should be undertaken even with the spontaneous nystagmus still to the bad side, provided that the hearing be entirely lost. By this early operation we may anticipate a number of cases which would develop and die of meningitis. Operation after the definite appearance of meningitis is at present an almost hopeless undertaking.

When complications are present the diagnosis may be more difficult, but it will readily occur that all the laboratory aids may be employed just as they are when no labyrinthitis exists. Thus if there be any reason to suspect a sinus thrombosis, blood culture as well as blood count may throw light on the condition. Lumbar puncture is also of great differential diagnostic importance.

When cerebellar abscess is present there may be persistent spontaneous rotatory nystagmus. Unlike that of vestibular origin the nystagmus remains to the affected side and increases in intensity from day to day.

The situation is different in the chronic diffuse cases. Many of them can give no definite history of the acute attack, many of them have been conscious of complete loss of hearing over a long period of years, and it is very evident that nature has successfully closed off the paths of intracranial infection. They suffer, however, from chronic otorrhoea and the possibility of extension must always be present. The writer confesses his inability to lay out for himself a definite procedure in these cases. Whether are the chances of the patient better if left alone or whether are the chances better if he be submitted to the labyrinth operation with the attendant risk of opening up the already sealed paths of infection. This would seem a most important point for dis-

cussion, and it is to be hoped that those who have operated on such cases will detail their experiences for the benefit of all.

The Neuman chart of the operative indications makes the chronic diffuse case one requiring immediate interference.

This chart, however, seems to be unduly complicated and not free from liability to criticism. The question of operation in any form of labyrinthitis can be simplified in this way. The labyrinthitis is diffuse or circumscribed. According to the Vienna School, all diffuse cases call for operation.

In the circumscribed cases the destruction is confined either to the cochlear nerve terminals or to the vestibular nerve terminals. In neither case, that is, as long as the disease remains circumscribed, is operation indicated.

But operation is indicated in either of these when we have evidence that the circumscribed labyrinthitis has become or is becoming diffuse. This extension will be disclosed by evidence of the irritation or destruction of the hitherto functionating end organ.

Thus if the disease has been confined to the vestibular apparatus, the onset of deafness will prove the extension to the cochlear apparatus. The disease being now diffuse, operation is indicated.

If, on the other hand, the disease has been confined to the cochlear apparatus, extension to the vestibular apparatus will be evidenced by the appearance of spontaneous nystagmus, nausea and vomiting and disturbances of equilibrium. Should this extension run the course previously described and the vestibular apparatus cease to functionate, the circumscribed labyrinthitis has become diffuse and operation is now indicated.

It does not seem necessary to complicate the operative indications by adding the results of the fistula test. When positive the only information of value with regard to the necessity of operation is that the vestibular apparatus is functionating. The mere fact of the presence of a fistula has no value either way in deciding for or against operation.

REPORT OF LABYRINTHINE CASES BEARING ON INDICATIONS FOR SURGICAL INTERFERENCE.

By NORVAL H. PIERCE, M. D., Chicago, Ill.

At the last Washington meeting of this society it may be remembered that I reported a series of labyrinthine cases together with the results of treatment. These were published in the Laryngoscope. In so new a subject as labyrinthine surgery I believe we should all bulletin our cases as soon as accurate conclusions may be drawn from them. In this way we may sooner reach permanent and accurate rules for our guidance. This is my excuse for presenting the following paper.

Case I. Male, aet 29, printer. Complains of suppuration from the right ear for many years. Three days before he came to me he was seized with an attack of vertigo and nausea, which continued for about 48 hours. At the time I saw him the vertigo had become less, but was still present to a slighter degree. Status praesens. Otoscopy, left ear: membrane cloudy; sunken; light reflex absent. Whisper for numerals 18 feet. Rinne—H (18/35). Weber to L. Schwabach—5″. Low limit G_2. High limit 2.5. Right ear: fundus obscured by thick foul discharge. Marginal perforation in superior posterior quadrant.

Tested with Neumann's hoise apparatus the right ear was found totally deaf to the Edelmann's tone series. There was marked spontaneous rotatory nystagmus to the left nearly approaching the second degree, but none to the affected (right) side.

Rotation to the right had little or no influence on the right nystagmus. Rotation to the left was negative. Caloric reaction of right was negative. Compressing the air in external auditory canal produced the long component to the left and aspiration reversed the movement. Temperature 99.6, pulse 88. The patient was put to bed with alcohol

dressing to ear. In a week the vertigo had practically entirely disappeared. But the right labyrinth did not respond to either caloric or rotation tests. On the other hand the fistula test was as marked as ever, producing violent vertigo. A radical mastoid operation was now performed. A most careful search with the aid of adrenalin failed to locate the fistula. There was none to be seen either in the horizontal canal or promontory, there was nothing unusual in the region of the stapes. The labyrinth was not touched. After the operation the patient did not complain of vertigo. The nystagmus gradually subsided, and he was discharged with a dry ear in about two months. No fistula symptom after six months. The ear remains totally deaf.

Case II. S. L., aet 26, tailor. Complains of vertigo, which prevents him from working. Has had similar attacks, but never so bad as the present. Has had a discharging left ear for years. Status praesens. Right ear negative. Left ear: Large perforation (marginal) in posterior portion. Thick foul pus, rather scant in quantity. Spontaneous nystagmus to both sides 1 deg. Caloric reaction negative. Turning to right negative, that is, does not increase nystagmus. Turning to left increases nystagmus. Hearing with a Koenig rod in right ear and with Neumann's apparatus, patient occasionally distinguishes word spoken in a loud voice. Fistula symptom marked. Compression causes violent nystagmus with long, slow component to the right. He had to hold on to chair to prevent falling. The patient was at once sent to the Illinois Eye and Ear Infirmary and kept in bed. The vertigo disappeared in two weeks, together with the spontaneous nystagmus to the left, that to the right remaining to a very slight degree. He became dissatisfied with the accommodations at the Infirmary and left. Several days afterwards he again appeared at my office, complaining of returning vertigo. The left ear at this time was entirely deaf and the spontaneous nystagmus to the right more marked, but there was none to the left. Fistula symptoms as before. He was sent to the pay patient department of Michael Reese Hospital, where I performed the radical mastoid operation

three days after his admittance. A slit with blackened edges was easily detected in horizontal canal. When pressed with the probe the eyes slowly moved to the right. The labyrinth was not operated on. Temperature before the operation was 98.5; pulse 76. After the operation it never ran above 100.4 on the third day. The mastoid wound healed favorably and was dry in five weeks. I kept him under observation for six months after the operation. The fistula symptom could be elicited for some time after the ear became dry, but it gradually disappeared. He did not complain of vertigo after the first week. The hearing improved so that he could hear whisper in the left ear for about 18 inches, and the rotary nystagmus could be produced to the left by rotation to the right of 6 seconds' duration. Warm caloric test of left ear produced nystagmus to the left.

Here we have two cases of diffuse serous secondary labyrinthitis which recovered after the radical mastoid operation without the labyrinth operation. In one case the auditory static function was restored to a moderate degree. In this case there was a demonstrable fistula. The time required for healing was not prolonged in either case. In conjunction with the other cases already published I believe I am in a position to formulate the following rule for our guidance in the treatment of these cases; that is, cases that show incomplete or complete loss of cochlear and static function, but with the fistula symptom still present, the patient should be placed at complete and continuous rest in bed during the active symptoms (vertigo, nausea, spontaneous nystagmus) unless there is sudden loss of the fistula symptom or there are symptoms indicating involvement of the cranial contents. In which event we may proceed with the radical labyrinth operation at once. In the absence of cranial symptoms we should postpone all operative procedures until the active labyrinthine symptoms have subsided. Then proceed with radical mastoid operation. A careful watchfulness should be maintained for meningeal symptoms. In case of fever above 101 F. with headache, stiff neck and increase in the cytologic contents of the cerebro-spinal fluid, together with positive

albumen test, the radical labyrinth operation should be performed at once. The operation performed at this time will give as great a protection as if performed at the time of the radical mastoid. If, however, the virulence of the infection is such as to cause a diffuse suppurative meningitis, the labyrinthine operation would have been useless in preventing the issue if performed at the time of the radical mastoid operation. On the other hand, if we are too precipitate with opening the labyrinth we may change a serous into a purulent condition, and by the trauma incident to our operative procedures force the infectious material through the perivascular spaces of the structures which traverse the internal auditory canal into the serous spaces of the meninges.

Case III. At the International Medical Congress at Buda Pesth I reported a case of cerebellar abscess of labyrinthine origin successfully operated on. The operation was performed in February, 1909, and the patient returned with the following symptoms in March, 1910. In the intervening period I had seen her several times. There was always nystagmus toward the left (healthy) side, but, no vertigo. The right cochlea did not react to either caloric or rotation tests. She returned because of a constant uncertainty of equilibration which had recently appeared and attacks of vertigo of such severity that she was unable to walk or stand. She had gained in weight and strength. Temperature was normal and pulse 82. There was a slight discharge from the operated ear from a fistula in the anterior inferior portion of the cavum. At this point there was some granulation tissue and the probe disclosed necrotic bone, but the balance of the cavity made by the radical mastoid operation was covered with dry epithelium. The ear was again tested and found to be absolutely deaf and did not respond to either the caloric, rotation or galvanic tests. But there was spontaneous mixed nystagmus to the left which constantly all but reached the second degree, and at times when her vertigo was most severe actually reached the third degree. The left ear was found to be normal for hearing (over 20 ft. for whisper) and for tuning fork tests, except that bone

conduction was slightly shortened and the upper tone limit slightly reduced (2.3 Edelmann galton whistle). Turning ten times to the left, that is, placing the left normal horizontal semi-circular canal in the so-called negative phase, increased the nystagmus to the third degree; while rotation to the right had little or no effect on the spontaneous left nystagmus. The patient was kept under observation. The vertigo gradually diminished and disappeared, but not the nystagmus. Six months after the patient had returned with the foregoing symptoms the left static apparatus responded normally to turning and caloric tests, but there was no compensation established.

The hypothetical explanation which I have worked out to explain this unusually interesting case is as follows:

Each crysta in each semicircular canal is composed of two physiological parts: one that when stimulated discharges major nervous impulses which produce the long component of a nystagmus to the opposite side; this is the apparatus which produces the phenomenon that results from a crysta when it is in what we have termed the state of highest physiological activity. The nerve fibres from this part of the crysta pass to the Deiter ganglion on the same side. The other physiological part produces impulse, which may be termed minor physiological impulses. The nerve tract from this part crosses to the opposite Deiter ganglion. These impulses, both major and minor, are produced whenever a crysta is stimulated, but the minor impulses are inhibited by a centre in communication with Deiter's ganglion of the corresponding side as the crysta from which the fibre originates. This inhibition of the crossed impulses occurs from Deiter's ganglion when the major impulses are being transferred through it to the nystagmus producing mechanism. Otherwise we would have no nystagmus from stimulation of a given crysta because both sides would be in balance. Suppose, now, the right labyrinth was destroyed, as in the case under consideration, and an irritation from inflammatory action or the contraction of scar tissue occurred in the

path of the crossing fibres in the region of their inhibiting ganglion. This irritation would be transmitted to the opposite Deiter's ganglion and we would have produced a nystagmus to the left.

ON THE ORIGIN OF COMPENSATORY TONUS AFTER DESTRUCTION OF THE LABYRINTH.

By GEO. E. SHAMBAUGH, M. D., Chicago, Ill.

In 1909 a young man consulted me because of the sudden loss of hearing. He was 27 years old and had suffered from chronic suppuration in both ears since childhood. Five years previous he had developed an acute mastoiditis on the left side, for the relief of which a mastoid operation had been performed. During this trouble he suffered for a time from vertigo and since then has not been able to hear with that ear. He came to me five or six days after the sudden loss of hearing in the right ear. For several months previous to this accident he had experienced from time to time attacks of vertigo (fistula). The loss of hearing in the right ear was associated with the development of pain in the ear and an increase in the purulent discharge. When I saw him he was complaining of severe headache and he had a temperature of 101°F. The patient stated that at no time after the sudden loss of hearing in the right ear had he experienced any vertigo. An examination by me failed to discover any evidence of spontaneous nystagmus, nor could any response be obtained either by the rotation or the caloric tests. There was no vestige of hearing in either ear. A diagnosis was made of destruction of the left labyrinth of five years' duration and of diffuse suppuration of the right labyrinth of one week's duration. Because of the fever and headache a radical mastoid operation and labyrinth exenteration was at once performed. A fistula was discovered in the horizontal canal and the labyrinth was found filled with pus. No labyrinth symptoms followed the operation and the patient made a good recovery.

Voss (1) reports a case where he had made a diagnosis of long standing destruction of one labyrinth (2) and where

(1) Wodurch entsteht der Nystagmus bei einseitiger Labyrinthverletzung? Verhandl. d. Deutsch. Otol. Ges. 1907.

(2) It is not clear from the report that this labyrinth was entirely dead.

an accidental opening was made in the labyrinth on the other side during a mastoid operation. There developed in this case a pronounced nystagmus to the opposite side, just as though the accident had occurred in a case where both labyrinths were normal. The spontaneous nystagmus in this case reported by Voss did not disappear completely until after several months.

Experiments on animals, where the second labyrinth is destroyed shortly after the disturbance of equilibrium that follows the loss of the first labyrinth has been readjusted, seem to show the symptoms of disturbed equilibrium which follow the loss of the first labyrinth.

The symptoms observed in these cases are of special interest in connection with the question of the origin of labyrinth tonus and particularly of the development of the compensatory tonus following the destruction of a labyrinth. To assume the existence of automatic tonus centers and to refer the explanation of the phenomena of labyrinth tonus to these centers is hardly a satisfactory answer to these questions, especially since there is no analogy in physiology for the existence of such centers and because all of these phenomena find a satisfactory explanation without these tonus centers.

Normally the voluntary muscles are under the influence of tonus impulses from the labyrinth. These impulses from the two labyrinths are equal though antagonistic and a state of equilibrium is sustained. The sudden cessation of tonus impulses from one labyrinth disturbs this equilibrium and a spontaneous nystagmus develops. It is well known that a loss of one labyrinth must take place rapidly in order to produce symptoms of disturbed equilibrium. Where the loss of function in the one labyrinth is gradual the process may go on to complete destruction of function without at any time giving rise to symptoms of disturbed equilibrium. Moreover it is known that in those cases where there has been a sudden destruction of function in one labyrinth the disturbance of equilibrium which is thus occasioned by the complete uni-

lateral suppression of tonus impulses from the labyrinth disappears after a shorter or longer interval.

The explanation for the readjustment which takes place in the disturbance of equilibrium following the unilateral destruction of one labyrinth seems quite clear. This readjustment is due to the development of compensatory tonus impulses to take the place of the tonus from the destroyed labyrinth. When the loss in function is slow enough the development of these compensatory impulses takes place hand in hand with the loss of the labyrinth tonus and no disturbance of equilibrium can develop.

Before we can understand the phenomena of labyrinth tonus it is necessary to have in mind certain facts in the physiology of the semi-circular canals. These are: first, that the hair cells on one side of a crista are stimulated by an endolymph current in one direction and the hair cells on the opposite side of the crista are stimulated by a current in the opposite direction; second, the hair cells on one side of a crista when stimulated produce nystagmus in the plane of its canal and directed towards one side, while the stimulation of the hair cells on the opposite side of the same crista produce nystagmus in the same plane but directed to the other side; third, the reaction following the stimulation of the hair cells on the two sides of each crista is unequal; fourth, in each canal the greater reaction follows the stimulation of those hair cells, impulses from which direct the nystagmus towards the same side. Tonus impulses from the labyrinth have their origin in the hair cells of the cristae. From each labyrinth therefore, arise tonus impulses for the muscles which direct nystagmus to the same side, as well as for the muscles which direct nystagmus to the opposite side. The stronger tonus impulses from a labyrinth are those which go to the muscles directing nystagmus to the same side, because, as just pointed out, the stimulation of the hair cells which give rise to these impulses produce a greater response than does the stimulation of those hair cells, impulses from which go to the muscles directing nystagmus to the opposite side. When the tonus impulses

from one labyrinth are suddenly suppressed the balance of equilibrium is disturbed and the tonus from the normal labyrinth, acting without the restraint of impulses from the opposite side, produce nystagmus directed towards the normal side.

What now is the origin of these compensatory tonus impulses that develop after the destruction of a labyrinth and which restore the disturbed balance in equilibrium? These compensatory impulses may have two sources. One source is the compensatory increase in those tonus impulses from the remaining labyrinth which direct nystagmus to the opposite side. Should restoration of equilibrium be due entirely to compensatory tonus from this source it would mean that the tonus impulses from the hair cells on the two sides of each crista in the remaining labyrinth are equal. This in turn should mean that a current of endolymph in one direction in a canal would produce a reaction just as great as a current in the opposite direction. This phenomena has been observed by Ruttin (1) in several cases where there had been a long standing destruction of one labyrinth. Here he observed that the nystagmus following rotation in one direction was just as great as that following rotation in the opposite direction. In cases of this sort, where the restoration of balance after the destruction of one labyrinth is due entirely to compensatory impulses from the opposite labyrinth, it is quite clear that in the event of the sudden destruction of the remaining labyrinth no pronounced disturbance of equilibrium and no nystagmus should follow, for the reason that the destruction of the second labyrinth would be the same as the simultaneous destruction of both labyrinths in its effect on tonus. It is possible that in the case reported in this article the absence of disturbed equilibrium after the loss of the second labyrinth is to be explained in this way.

In most of the cases where a restoration of equilibrium has been accomplished after the loss of one labyrinth it is evident that this is not due entirely to the compensatory

(1) Verhandlungen der Deutschen Otologischen Gesellschaft. Juni, 1911, p. 212.

increase in tonus from the opposite labyrinth, because in applying the rotation test in these cases the after nystagmus to the normal side is found to be much stronger than to the defective side. This test shows that the reaction following stimulation of the hair cells on the two sides of the crista is still unequal. It is clear, therefore, that in most cases the restoration of equilibrium after a unilateral labyrinth destruction is accomplished by a compensatory tonus which, in part at least, is developed independent of the opposite labyrinth. It is even possible that in some cases the restoration of equilibrium is accomplished entirely through the development of this extra labyrinth compensatory tonus. I cannot accept the view that there exist automatic tonus centers in Deiter's nucleus, where tonus impulses are stored or originated. The tonus impulses which emanate from Deiter's nucleus have their origin in afferent impulses from the labyrinth and elsewhere. These impulses are in a measure modified in their passage through Deiter's nucleus, but tonus impulses are neither stored nor developed automatically here.

It is known that afferent impulses other than those from the labyrinth convey tonus to the voluntary muscles. The tonus impulses from the labyrinth are probably the more important as they are the more delicate. The extra labyrinth tonus impulses are the cruder and more primitive. Those from the labyrinth come from a highly specialized mechanism developed for the particular purpose of preserving equilibrium. A sudden loss of the labyrinth destroys not only the labyrinth tonus from this side, but the sudden shock to the center in Deiter's nucleus destroys temporarily much of the extra-labyrinth tonus. This it does by a process which Monakow (1) has designated as diaschisis. The rapid recovery of the disturbed equilibrium following the loss of a labyrinth is to be explained through the action of the extra labyrinth tonus. The first process in the recovery of equilibrium is the restoration of the normal extra labyrinth tonus through the disappearance of

(1) Zeitschrift f. Psychologie, 1909, Bd. 54.

the suppression of this tonus by the action of diaschisis. But this restoration of the normal extra labyrinth tonus is not sufficient to restore the disturbed equilibrium arising from the unilateral loss of labyrinth tonus. Its place must be supplied by the development of an additional tonus. This additional compensatory tonus, which early takes the place of the tonus from the destroyed labyrinth, seems to develop chiefly from the extra labyrinth impulses. This is shown by the fact that the response of the hair cells on the two sides of the cristae in the remaining labyrinth continues to be unequal. In applying the rotation test the after nystagmus to the normal side is greater than to the opposite side. It appears to be only in cases of long standing destruction of one labyrinth where the compensatory tonus from the remaining labyrinth supplants completely this compensatory extra labyrinth tonus. In these cases the response of the hair cells on the two sides of the cristae in the remaining labyrinth is the same, and in the rotary test the after nystagmus to the defective side is as strong as towards the normal side.

It is evident that these extra labyrinth compensatory tonus impulses which develop after the destruction of one labyrinth will tend to balance the impulses from the several cristae of the remaining labyrinth. In case of the sudden destruction, therefore, of the second labyrinth, a disturbance of equilibrium and a nystagmus should develop exactly as though the function of the first labyrinth had been restored. The nystagmus will be directed to this side. The disturbed equilibrium and spontaneous nystagmus which follows the sudden destruction of the second labyrinth are produced by the compensatory extra labyrinth tonus, which had developed to take the place of the tonus from the labyrinth first destroyed, acting now without the restraint of tonus impulses from the labyrinth last destroyed. The restoration of equilibrium and disappearance of the spontaneous nystagmus which follows the destruction of the second labyrinth, is dependent entirely on the development

of extra labyrinth tonus to take the place of the tonus from this labyrinth.

The restoration of equilibrium which follows the destruction of both labyrinths is never a complete restoration of the normal equilibrium. The extra labyrinth tonus which supplants the labyrinth tonus is not as perfect a mechanism for preserving the equilibrium as in the tonus from the highly specialized sense organs in the labyrinth. In the case of the sudden loss of one labyrinth the rapid readjustment of equilibrium which follows is dependent, as pointed out above, on the development of the extra labyrinth compensatory tonus. The readjustment is not a complete substitute for the labyrinth tonus. It is only in cases of long standing unilateral labyrinth destruction where the restoration of equilibrium is apparently perfect, and here the compensatory tonus from the opposite labyrinth has fully developed so as to supplant completely the compensatory extra labyrinth tonus.

Discussion.

Dr. B. Alexander Randall, of Philadelphia, thought it should not be forgotten that man is a three-footed creature in respect to equilibrium. He was glad Dr. Shambaugh laid stress upon this extra-labyrinthine matter. From the cerebellum was derived, in large measure, the ability to guide one's self and one's relation in space. A person might lose both labyrinths and with the eyes and cerebellum make fair locomotion out of the water. Streeter's experiments with the tadpole were interesting in this connection. With the labyrinth removed or transposed the tadpole which in water could have only slight muscular sense, could not swim at all right side up. Streeter had not extended his work to the frog.

In a case such as that reported by Dr. Pierce one must consider whether the changes noted are not due to a cicatricial condition in the labyrinth. Cerebellar abcess might cause the trouble. Was not Dr. Shambaugh rather "splitting hairs" with reference to the two-sidedness of the crista

vestibuli? It reminded him of Gould's view of the astigmatic eye, with retinal cones ambliopic north and south, but not east and west.

Dr. Emil Gruening, of New York City, emphasized the importance of the practical questions which had been raised by the readers of the papers. While the work might seem hypothetical it had its practical phases. Dr. Pierce is conservative and still holds views which were held before the labyrinth was so frequently operated upon. It had long been assumed that in the presence of suppurative otitis media, there might likewise be vestibular or cochlear disturbance, with production of exudate by a chemotaetic process. In former times all these symptoms were observed and it was known that they would disappear without operation upon the labyrinth. What was true formerly was true now. He would have proceeded just as did Dr. Pierce, —operate from the middle ear, and clear out the diseased area, so that there would be no further action on the labyrinth by chemotaxis.

Dr. Welty of San Francisco. I think the paper of Dr. Rae is more in line with my theory for operative indications than any that I have heard at this meeting or that of the American Medical Association. At the same time, I don't think he goes far enough at that. I consider the Neuman classification for operative indications, the most careful and systematic that we have at present. When one attempts to change the indications for operative interference, they are more likely to do harm than good. If Dr. Canfield's observations are correct, when added to the Neuman classification will give you an operative chart beyond question. I must admit that some of the cases that fall in the operative class get entirely well without operation. Now, this is not entirely confined to one particular class, but is almost equally divided. I speak from my experience of four cases that naturally fell in the operative class, but were not operated. All these cases recovered. One continues to have a discharge from the ear. On the other hand, I have operated eight cases; they are all

well and free from discharge. Two had a temporary facial paralysis. Of the operated cases, I will say that they were all improved almost immediately with the exception of one who had an incomplete labyrinth operation. Very much like Dr. Pierce's case. This disturbance of equilibrium and vertigo are directly traceable to the incomplete labyrinth operation. This was particularly called to my attention in Vienna some three years ago, for they had a similar experience there. However, this happened only when such surgeons operated who did not believe in the complete destruction of the labyrinth, which included the internal auditory meatus.

I understand from a recent publication signed by Professor Urbantchits, that when there is hearing or remnants of hearing, they do not operate. While this statement seems quite rational, I cannot agree with the author until he can demonstrate to me beyond a question of doubt, how he can exclude a pus infection of the vestibular apparatus. Until such a time, I shall consider it a serious mistake to do otherwise than operate. If you reason from a pathological standpoint, you cannot come to any other conclusion. When a labyrinth operation is to be done, the only one to be thought of is the Neuman, because it is surgical and complete. On the other hand, incomplete operations are made and for some reason or other you wish to make it more complete, you invariably fracture the petrous portion of the temporal bone, which is a serious accident in the midst of an infected field.

Dr. Wendell C. Phillips, of New York City, said it was to be hoped that otologists would continue to report cases of acute labyrinthitis until some course of procedure could be agreed upon. He cited two cases. The first patient was a woman, about thirty years of age, who had had chronic suppuration of the middle ear for some years. For three or four weeks previous to admission to his clinic at the New York Post-Graduate Medical School and Hospital she had had attacks of vertigo and vomiting, with some fever, according to the family. When he saw her she gave evidences

of labyrinthitis existent for some time and the tests indicated that she had fistula. She did not have high temperature, and the cerebrospinal fluid showed no evidence of infection. Radical mastoid operation revealed cholesteatoma and a fistula of the external semicular canal. She made a complete recovery.

In the second case the patient was a young Greek, twenty-three years old, of marvellous physique, who had had acute suppuration of the middle ear for two months. He had exhibited mastoid symptoms, and had been examined by various men, and was under the care of one of his assistants. He was taken to the Manhattan Eye, Ear and Throat Hos- Finally he developed attacks of acute vertigo and vomiting. pital, and placed in a ward, where he saw him. The patient presented all the symptoms of acute diffuse labyrinthitis, without any cerebral symptoms whatever. The speaker was about to perform the radical mastoid operation, when the man developed suddenly a high temperature. The cerebrospinal fluid was drawn off, examined, and streptococcus infection found. He operated and found a very extensive infection. As soon as the labyrinth operation was completed, knowing that there was infection of the meninges, drainage of the cisterna magna was performed. The patient had shown absolutely no mental symptoms, his mind being perfectly clear before and after the operation. With the drainage which was obtained the man lived nine days. There were never any signs of intracranial pressure, the history being that of a person with an acute infection. The temperature never ran above 102 and 103 degrees Fahrenheit. On the eighth day after the drainage operation he showed some signs of weakening; on the ninth day he asked for a drink of water, sat up, drank the water, and fell over dead. The drainage prevented intracranial pressure, death occuring from infection.

Dr. John F. Barnhill, of Indianapolis, Ind., said his experience had coincided with that of Dr. Gruening. He had had very little experience with operation upon the labyrinth, because he had recognized very little necessity for it.

Patients with symptoms of labyrinthitis had gotten along very well with operations such as that mentioned by Dr. Gruening. He could recall no instance of death in his practice from failure to open the labyrinth.

Dr. Lewis, of Dubuque, Ia., asked Dr. Shambaugh if galvanic examination had been made of the rare case in which had been observed, studied, and reported the establishment of compensatory extra-labyrinthine tonus after total destruction of one labyrinth. Whereas in a normal individual, or in a person lacking one labyrinth in whom such compensatory extra-labyrinthine tonus had not been established, galvanic stimulation *will cause nystagmus,* in a case such as the one described, with fully established extra-labyrinthine tonic compensation, the application of galvanic stimulation must obviously be *powerless to elicit nystagmus reaction.* Should actual experiment upon such a case demonstrate *no nystagmus response to galvanic stimulation,* it would establish the proof of the logical and very plausible theory expounded by Dr. Shambaugh.

Dr. Rae, in closing the discussion, said that he thought it to be of the utmost importance to arrive at some definite method of determining the operative indications. The classification of Neuman could be simplified. The column in his table with reference to the fistula test could be omitted with advantage. A positive fistula test informs us of the presence of the fistula and *that the vestibular apparatus on that side is functionating.* The important point with reference to operative indications is not the presence of a fistula, but the fact that the vestibular apparatus is functionating. This point is disposed of in another column of Neuman's classification.

Many of the cases reported show a positive fistula test with a non-functionating vestibular apparatus. This is obviously an error. Neuman's table is not free from the same mistake.

With regard to Dr. Whiting's remarks, Dr. Rae did not by any means take the position which Dr. Whiting had assumed him to take. He had meant to emphasize the importance in

the presence of an acute purulent labyrinthits of the early recognition of the onset on cerebral complications.

Dr. Pierce, in closing the discussion, said he had reviewed cases, as many as would report back to him, who had undergone operation several years ago. In some of the cases, operated five or six years ago, there was labyrinthine involvement. It was a significant fact that the majority of these cases had done very well, although labyrinthine involvement had not been recognized at the time of operation. The labyrinth had not been operated on. The cases which had eventuated in meningitis from radical mastoid operations, had been very few indeed. He believed nature has a very strong tendency to block off labyrinthine inflammation, even in purulent cases. He cited a case in point. A woman, with suppurative otitis media, with cholesteatoma, had been operated upon not by the radical labyrinth operation, but simply enlarging the fistula of the semi-circular canal and taking away the promontory. She returned some years afterward with violent nystagmus to the opposite side, and with a running ear, the latter of unknown duration, probably ever since the operation. Examination revealed a sequistrua. The fistula was enlarged and a large sequestrum, probably involving the entire labyrinth, was removed. Facial paralysis had followed the original operation. In this case there was extensive labyrinthitis without meningitis. While some cases demand radical labyrinthine operation, he believed these to be relatively few. He agreed with Dr. Rae concerning the impossibility of differentiating between diffuse serous secondary labyrinthitis and diffuse purulent labyrinthitis in some cases. In each there might be loss of cochlear function, the static function, and the negative fistula test. The main indication for operation being the development of cerebral symptoms, especially meningitis, in many cases these symptoms should be detected at the earliest moment.

It was important to operate upon the cases at the very onset of the intracranial involvement. To this end the cerebrospinal fluid should be subjected to chemical,

cytologue and bacterial examinations before the onset of gross clinical symptoms. In other words, the only adequate and reliable method of procedure is to take into consideration the whole picture, the results of labyrinthine tests and the condition of the cerebrospinal fluid.

Dr. Shambaugh, in closing the discussion, said that he did not quite understand the point made by Dr. Rae regarding the absence of bone conduction on the side where the labyrinth was destroyed. In cases of unilateral destruction of the labyrinth bone conduction should still be present, and it may or may not be lateralized to the normal side. It is not easy to make a definite diagnosis of destruction of function in one ear. With the use of the Bárány noise apparatus in cases where there is still a vestige of hearing this may be obscured so that the patient apparently hears nothing with the affected ear. It is not always possible to make a differentiation clinically between serous labyrinthitis with suppression of function and suppurative labyrinthitis with destruction. In serous labyrinthitis during the milder stages, that is, during the onset as well as during the stages of recovery, where congestion is the chief phenomena in the labyrinthitis, spontaneous nystagmus is often directed toward the affected side, but during the heighth of a severe serous labyrinthitis there may be present a complete suppression of function with the nystagmus now directed towards the normal side, just as though there was present a diffuse suppuration of the labyrinth. Dr. Shambaugh knows of no positive way of making a differentiation clinically between these two types of processes, the serous and the suppurative. For this reason he is inclined to be very conservative about operating upon the labyrinth in the presence of an acute labyrinthitis. The mere presence of an apparent loss of function is not sufficient, in his opinion, to warrant an operation upon the labyrinth. He has always waited for the development of more severe symptoms, such as increased headache and rising temperature, as suggested by Dr. Rae. He recalls but five cases of this sort where he has felt justified in operating on

the labyrinth. In all of these cases the patient made a good recovery after thorough drainage of the labyrinth.

Replying to the question of Dr. Lewis regarding the application of galvanism in cases of destruction of one labyrinth, the speaker stated that he had not carried out these tests. He would expect, however, that in cases of unilateral destruction of the labyrinth where the restoration of equilibrium is brought about through the compensatory development of extra-labyrinth tonus, that the application of the cathodal current to the normal ear would produce nystagmus directed toward this side. On the other hand, he would expect in cases of long standing unilateral destruction, where the restoration of equilibrium had been brought about by the compensatory increase in tonus from the less sensitive hair cells of the cristae in the remaining labyrinth, that the application of the galvinism might not be able to produce any nystagmus. The reason being that the reaction following stimulation of the hair cells on the two sides of the cristae in the normal labyrinth produces an equal response, that is, affects equally the muscles that direct nystagmus toward the defective side as well as towards the normal side.

Dr. Randall suggested that the point made by the speaker regarding the stimulation of the hair cells on one side of the crista by a current in one direction and of the hair cells on the other side of the same crista by a current in the opposite direction, was "hair-splitting." The speaker pointed out that this is the view now generally accepted since the more recent work by Breuer.

A CASE OF PURULENT LABYRINTHITIS FOUND AT AUTOPSY.

By W. SOHIER BRYANT, A. M., M. D., New York.

Patient, a woman 60 years of age. Death from pneumonia. Autopsy showed lobor and fibrous pneumonia chronic purulent otitis media (left), and purulent labyrinthitis without involvement of the meninges. The labyrinthitis was not suspected during life.

The findings in this case show that purulent labyrinthitis may be present without intra-cranial complications, and that when complicated by other serious lesions, purulent labyrinthitis may be overlooked. This case also shows that, in cases of chronic purulent otitis media, terminal thoracic complications are often dependent upon and secondary to, the chronic purulent otitis media.

During the last two years the author has seen five autopsies on cases of terminal pneumonia, secondary to chronic purulent otitis media.

AN EXHIBITION OF RADIOGRAPHS OF THE MASTOID PROCESS SHOWING THE NORMAL AND PATHOLOGICAL CONDITIONS.

By WENDELL C. PHILLIPS, M. D., New York, N. Y.

Dr. Wendell C. Phillips, of New York City, presented a series of radiographs of the mastoid process, showing normal and pathological conditions. Each radiograph was explained and commented upon by the speaker as follows:

1. External auditory canal.
2. Lateral sinus.
3. Outlines of auricle.
4. Entrance to mastoid emissary vein.
5. The condyle of lower jaw.

Twelve negatives were shown and described. The special anatomical points were indicated upon the plates as follows:

Case 1—M. I. A normal left mastoid process showing malleus.

Case 2—H. O. M. S. C. Sclerotic mastoid right side, normal mastoid left. In X-ray plate it was shown that there was small amount of pus in the attic region. Radical mastoid operation confirmed this and showed softened bone in the attic region where the dura was exposed. The tip cells and deep cells along the canal wall were congested and softened.

Case 3—Phlebetis of lateral sinus. O. M. S. C.

A cholesteatoma was suspected. Operation postponed for twenty four hours and X-ray taken, the idea being to get a good plate to show sclerotic bone and possibly cholesteatoma. *Operative findings*—Cholesteatoma, abscess of sinus with pus oozing from anterior wall of sinus. The portion of sinus affected is clearly shown in the negative by its cloudy appearance.

Case 4—P. Q. age 46 years. Acute suppurative otitis media, with mastoid tenderness. Paracentesis performed and case cleared up. No operation. The plate shows a mild inflammation of the mastoid cells, right side. Left side normal.

Case 5—R. L. Slight inflammatory condition of cells of the left mastoid. No pus in cells. Not an operative case.

Case 6—S. K. Acute mastoid with the classical symptoms. Right mastoid showed evidence of inflammation in the cells, but the walls are still intact and there is no pus. At the mastoid operation, the whole mastoid tip was blue and discolored. Two or three cells in the tip contained pus. It was a case with an extensive mastoid just beginning to break down.

Case 7—M. T. Left side pneumatic and normal. Right side diseased. Operative findings: Diagnosis confirmed by operation.

Case 8—J. K., age 37. Epidural abscess. Left side normal, small mastoid. Right side badly involved. Operation showed a large epidural abscess (shows as a thinning of the bone). Pus fills the mastoid destroying the cells except in the upper part of the antrum where some of the partitions are preserved.

Case 9—F. W. Simple mastoid operation. History: Purulent discharge off and on for eight years. Had lues eight years ago. One month ago discharge started again with severe pain. Three days ago a large swelling appeared behind the right ear. Suffered from tinnitus and some vertigo.

Operative Findings: Edema of the neck and perforation of the cortex one inch above the tip. The upper part of the mastoid was eburnized. Lower part of the mastoid cellular. The mastoid cells are full of pus.

Case 10—A. D. Sclerotic mastoid. Examination of the patient shows middle ear cavity epidermicized. Total defect of the drum. History: O. M. S. C. for two years. Discharge has now ceased, but the patient has slight vertigo.

X-ray shows sclerotic mastoid in the posterior part. The sinus and emissary vein can be seen. No operation.

Case 11—C. S. Three exposures. Simple mastoid operation.

IA—Left mastoid shows evidence of thin pus. The cells have not yet broken down. Accumulation of pus around canal.

IB—Plate five days later shows evidence of clearing up. The spot of pus about the canal has disappeared.

IC—Third plate shows evidence of process increasing. The cells are not as clear as in the previous plate. A fourth plate was taken later on which showed a further increase in the inflammatory process, but that plate has been lost. Very profuse discharge continued, and a simple mastoid operation was performed. Operative findings:—A very extensive mastoid, all the cells containing muco-pus. Part of the descending limb of the sinus exposed.

Case 12—H., age 36. History: Ear trouble began with an attack of coryza, three weeks ago. Discharge and pain in the ear three weeks. There was a swelling over the mastoid, very tender to the touch. Operative findings:—Cortex is perforated over the antrum. Cellular mastoid full of pus. Simple mastoid operation performed.

Discussion.

Dr. Herbert C. Birkett, of Montreal, Canada, said he had been engaged in similar work for the past five years, during which time he had obtained a large number of plates, and had acquired a certain amount of facility in reading them. He was convinced that radiography gives valuable aid to diagnosis in disease of the mastoid. He congratulated Dr. Phillips upon securing a plate which showed thrombosis in the lateral sinus. The speaker had one such plate in his collection. The presence of pus in the thrombus caused it to be shown.

Dr. S. MacCuen Smith, of Philadelphia, believed it required not only an expert to take plates like those Dr.

Phillips had presented, but one equally skilled to interpret them. Some gave valuable data, especially in locating the lateral sinus. By this means we could definitely determine whether the sinus was superficial or located very far forward.

Dr. B. Alexander Randall, of Philadelphia, said he had had much experience in this line. He cited one case of exostosis in which the radiographer pointed out to him upon the plate the seat of the exostosis—which he would have been very glad to know before operation.

Dr. Phillips, in closing the discussion, said a simple exposure was of less than thirty seconds' duration, and he had been informed that so short an exposure to the X-Ray could not cause serious harm.

HISTORY OF A CASE OF CHOLESTEATOMA OF THE EAR.

By E. GRUENING, M. D., New York, N. Y.

The patient of whom I wish to speak was under my observation 30 years. She was a woman who came to me when she was 24 years of age. In her infancy and youth she had often been troubled with discharge from the right ear. At the time of the first examination, 30 years ago, the drumhead did not exist; there was neither a malleus nor an incus, the tympanic cavity was dry and epidermized, the aditus and antrum communicated with the middle ear as freely as if a radical operation had been performed. No actual operation had, however, been done. The process of disease had formed a spacious cavity from which now and then cholesteatomatous masses descended into the external auditory canal. The ear was absolutely deaf. The patient would at times, especially after immersion of the head in a warm bath, complain of dizziness. I ascribed this symptom to the swelling of the cholesteatoma from imbitition of water, and treated the ear, when these symptoms appeared, by the instillation of glycerine and alcohol. After a few days of such treatment the cholesteatomatous material became dry, and the head symptoms would then disappear. When the attention of the patient was drawn to the circumstance that the water-logged condition of the cholesteatoma might cause the head symptoms, she refrained from filling the ear with water and the symptoms mentioned appeared but rarely.

During this long period of observation she was also seen, during her trips to Europe, by Politzer of Vienna, Betzold of Munich, and other noted aurists. None of these men proposed an operation, and all agreed that under the given conditions the treatment which she followed under my direction was sufficient.

In the month of March of this year she began to suffer from copious uterine hemorrhages. A gynecologist who was consulted found that she suffered from uterine fibroids and advised that the uterus be removed, but also stated that an immediate operation was not imperative.

On March 23d of this year she was suddenly taken with a severe chill, high temperature, (104.6), and frequent vomiting. The family physician who knew that she had been under my observation for her aural condition, considered the possibility of a systemic infection by way of the lateral sinus and desired to meet me in consultation. I saw her in the evening of that day. While I was in her house she had a severe chill and vomited. The temperature had risen to 105. The examination of the ear showed what I had seen before, viz., a perfectly dry middle ear, no tenderness of the mastoid and post-mastoid regions, no enlarged glands and no sensitiveness along the course of jugular vein. The ophthalmoscopic examination was negative, and I came to the conclusion that the sepsis from which the patient suffered was not caused by the condition of the ear. In order to observe the patient more fully and have the necessary laboratory tests made, she was transferred to a general hospital that very evening. The leucocyte count was 24,400 with 88% polynuclears. Enough blood was taken from the veins of the arm to make a blood culture. The gynecologist who had seen her previously re-examined her at the hospital that evening, and decided that the sepsis was not due to the condition of the uterus. He found, however, some tenderness of one ovary and ordered the application of an ice bag. During the night the patient vomited frequently, her temperature remained high, and she had several severe chills. Her mind remained clear. On the following morning, March 24th, I met Dr. Whiting in consultation, who corroborated my view of the case. One of the gynecologists of the hospital who was called in consultation disagreed with the previous finding in regard to the uterus and proposed an immediate panhysterectomy. The husband of the patient consented. I was present at the operation. In cutting through the right ovarian vein a small

amount of pus escaped. The removed uterus contained several submucous and intramural fibroids. The centres of some of the fibroids were softened and discolored. The patient rallied from this operation, but the temperature rose to 105.6 and the chills and vomiting continued. On the morning of the 25th of March we received the laboratory report of the blood culture, which had been made under Dr. Libman's supervision. The streptococcus was found in the blood. A medical consultant who was called by the family, advised an exploration of the right lateral sinus and ligation of the jugular vein. The patient had 40 respirations per minute, pulse 140, and in view of the fact that the haemoglobin was 60%, he thought she would bear the operation well. In order to gain time, a surgeon ligated the jugular vein while I laid bare the sinus. Though the operation lasted only a few minutes, the patient collapsed, but rallied after an intravenous infusion of hot saline solution. No thrombus was found in the sinus. The patient died that evening. Dr. Libman made a local post-mortem examination of the region of the lateral sinus, but found no clot. The pathologist of the hospital examined the uterus microscopically and found purulent thrombi in the uterine veins. This case demonstrates that though a chronic disease of the middle ear may be associated with a systemic infection and a positive blood culture, we are not entitled, in the absence of local symptoms, to assume that the diseased ear must necessarily be the causative factor. We should exclude all other possible foci of infection. The result of the post-mortem inspection of the sinus and the microscopic examination of the uterus showed that the operative procedure on the sinus was unnecessary.

Discussion.

Dr. B. Alexander Randall, of Philadelphia, had been radical enough to teach that practically all cases of chronic suppuration are cholesteatoma cases. There was a good deal of room for difference of opinion as to what constitutes cholesteatoma. Unquestionably in all cases of chronic sup-

puration there was some involvement due to the grafting of epiderm from the external auditory canal and the drum-head upon the mucosa, with proliferation of the mucosa; there was dermatitis, also, just as in the case reported by Dr. Gruening. It was seen more often in cases which have been radically eviscerated, according to the custom of his New York colleagues, than in cases operated upon by the less radical method which he practised. The case was fortunate indeed, which escaped it after such radical operation. He had seen cases repeatedly in which there was recurrence of the suppurative process, and even of cholesteatoma. In such cases as that reported there was free access to the antrum and cells, but he gravely questioned whether that case needed operation. From the history he believed the view taken a little too radical, and that the cholesteatoma was not properly suspected to produce a sinus lesion or infection elsewhere. Cholesteatoma might cause death from hemorrhage or might destroy the labyrinth, and, as tumors of the brain, might cause pressure upon the brain almost as much as malignant tumors. These, however, were neglected cases. He would not have operated upon Dr. Gruening's case.

Dr. George E. Shambaugh, of Chicago, asked whether post mortem examination of the bulb of the jugular had been made. He cited a case presenting chills and fever, of septic origin, in a patient who had had acute otitis media with a mastoid abscess several years before. No evidence of disease could be found in the region of the ear by external examination, but, as there was noting pointing towards any other cause for sepsis the speaker suggested that the cause might be found in an involvement of the sinus. The ear was opened, the sinus extensively explored but was found to be normal. A week later an abscess of the bulb discharged spontaneously through the lower part of the mastoid opening and the patient recovered. In another case presenting evidences of a septic involvement an operation was performed which disclosed a cholesteatoma in the antrum. The lateral sinus was normal but a septic abscess

was found in the bulb of the jugular. This condition was complicated the day following the operation by the development of a peritonsillar abscess which on opening discharged the same foetid pus which had been found in the bulb.

Dr. S. MacCuen Smith, of Philadelphia, said for many years it had seemed to be almost the consensus of opinion that the patient's interest was best conserved by operation for cholesteatoma which did not improve under other measures. Dr. Day's case was one in point. So long as cholesteatoma existed, the patient was subject to various complications. He referred to a paper read by him before the New York Academy of Medicine, in which he cited three cases of metastatic abscess of the liver traceable to aural suppuration. He did not agree, therefore, with Dr. Randall in this respect. While many cases might remain quiescent for years, yet with cholesteatoma present, the patient was subject to metastatic recurrences elsewhere, as well as intracranial complications.

Dr. Max Goldstein, of St. Louis, said there was still some vague points concerning cholesteatoma. Might the case reported by Dr. Gruening be considered as circumstantial evidence as to the connection between cholesteatoma and uterine fibroids, or is there some factor conducive to a corelation between cholesteatoma and other pathological conditions?

Dr. James F. McKernon, of New York City, agreed with Dr. Whiting. The best interest of the patient was conserved by removing the cholesteatoma, thus placing the individual on a safe basis for the future. To leave a patient with cholesteatoma was running a risk which no otologist should take.

Dr. Wendell C. Phillips, of New York City, agreed with Dr. McKernon. Every case in which cholesteatoma was demonstrated should be subjected to radical mastoid operation.

Dr. Thomas Hubbard, of Toledo, Ohio, asked Dr. Gruening concerning the problem of dealing with cholesteatoma when complicated by the fact that both ears are involved. Many of these patients have fair hearing and the ultimate issue seems to be deafness or extension of disease process. The majority of patients will take the risk of the disease to retain a useful degree of hearing even past the danger line.

Dr. H. O. Reik, of Baltimore, Md., said the majority of the speakers, in discussing this paper, seem to have assumed that there was a definite relationship between the existence of cholesteatoma in the middle ear and the diseased condition from which the patient died. He would like to ask the author of the paper if that was his view, because he did not understand him so.

Dr. Gruening, in closing the discussion, said the radical operation had been performed by nature in the case cited. There was no discharge of pus at any time. If he had been obliged to operate upon this patient he would have been forced to establish a permanent opening upon the outside, which would have been a disfigurement. That the gynecologist did not make the diagnosis should not cause the otologist to operate upon a patient for cholesteatoma. The case cited was one of the cases of cholesteatoma in which an operation was not necessary. In this he was supported by Politzer, Bezoid, and others who saw the patient. He would operate for cholesteatoma where there was any retention of material. If, in a given case of ear disease, streptococcemia was found, it was not yet demonstrated that the ear disease and the streptococcemia stand in the relation of cause and effect. The case here recorded demonstrates that a patient suffering from cholesteatoma of the ear and streptococcemia may have at the same time in some other organ a local process causing the bacterial invasion.

THE REPORT OF A CASE OF SINUS THROMBOSIS, EXCISION OF THE INTERNAL JUGULAR, WITH RECOVERY.

By CHARLES W. RICHARDSON, M. D., Washington, D. C.

On June 8, 1911, I was called to see a young woman twenty years of age, with the following history: Since her tenth year she had had a suppurative otitis, involving the left ear, secondary to an infection from measles. The ear had continued to suppurate moderately until the advent of the present illness. Until that time the ear had caused her no pain or discomfort other than the continuous discharge, which was always more or less offensive. She had been quite ill for over a week when I first saw her. She had had a severe chill, followed by profuse sweating for each of the five days preceding my visit. There was great spontaneous pain over the mastoid region, radiating over the head, more intense at night during all this period, increasing in severity each day. Digital exploration proved the whole mastoid region exquisitely sensitive. No temperature had been taken until the morning of the day when I was called into consultation by Dr. Billard, although, up until that morning, when Dr. Billard was asked to take charge of the case, the patient had been under the observation of an aurist. Dr. Billard's first clinical observation of the case caused him at once to recognize its nature and gravity. The temperature then taken gave a reading of 105. The patient was extremely anxious, suffering great pain, of a sallow complexion, with a temperature of 102, and a pulse of 120, when I saw her late the same evening. The auditory canal contained a small amount of extremely fetid pus. The cavity of the tympanum was completely exposed, the membrane and ossicle being entirely absent. A diagnosis of sinus thrombosis was made and the patient was sent immediately to the hospital for an operation early the next day. Blood count demonstrated a leucocytosis of 23,000.

The Operation. I had decided to open the mastoid first and then to be governed in my further operative procedure by the condition presented. The patient was in fair condition, with a temperature of 103, just before the administration of the anesthetic. On removing the cortex, over the antrum, extremely offensive pus actually projected itself several inches about the operative field. The sinus, which was unusually high and anteriorly placed, immediately filled in the place left by the escaped pus, embarrassing all further operative procedure until the sinus was dealt with. A free exposure was now made of the sinus, which was firm and hard throughout the whole limit of exposure. On opening the sinus it was found to contain an organized clot, which, on removal, measured three centimeters in length. As an evidence of the nature of the clot, through a mistake of the pathologist to whom it was sent to ascertain the form of infecting organism, it was mounted and cut, through the impression that it was a piece of tissue. The circulation was restored posteriorly through the removal of the clot. Free curatting into the jugular bulb failed to restore the circulation from below. The antrum and aditus were thoroughly curetted after the sinus had been given attention. The internal jugular was then exposed and excised throughout nearly its whole course. The jugular wound was closed and a gauze drain inserted. The patient was returned to her room in excellent condition. The highest temperature reached the following day was 100. The temperature never rose above the normal afterwards, and the patient made a perfectly uneventful recovery. The jugular wound healed throughout nearly its whole course by primary union.

Unfortunately, the infecting organism was not ascertained, through the mistake made in the instructions given to the pathologist. This case proved very instructive and interesting to us. as from the duration of the infection and the intensity of the symptoms, it seemed as though it were the most unfavorable case for successful operative intervention that we had met with; nevertheless, the result obtained proved contrary to our expectation.

A CASE OF SEPTIC SIGMOID SINUS THROMBOSIS, OCCURRING, WITHOUT PHYSICAL SIGNS, IN AN INFANT; OPERATION FOR LIGATION AND EXCISION OF THE INTERNAL JUGULAR VEIN.

By ROBERT LEWIS, M. D., New York, N. Y.

On February 26th, 1912, I was called to see an infant two years and four months of age, who had been suffering for about eighteen days from an acute otitis media of an apparently mild type. On examination, I found a very slight mucoid discharge from the left ear; the drum membrane was slightly œdematous; there was no prolapse of the posterior superior cutaneous wall of the canal, nor was there any stenosis of the canal; in a word, the picture presented was that of a very mild and subsiding acute otitis media.

On the right side, the drum membrane was found to be intact, but it was slightly congested and there was appreciable bulging limited to the superior and posterior third of the membrane. From the conditions observed in the two ears I was disposed to make a diagnosis of a very mild catarrhal inflammation of both middle ears—a condition that scarcely called for a resort to paracentesis of the drum membranes; but, because of the elevated temperature, 105° F., I thought it was safer to perform a double myringotomy.

On the following morning, Dr. J. J. Henna, the child's physician, saw the patient and telephoned me that the temperature had again risen to 105° F. It was then thought best to send the child to the hospital. I saw the patient at the New York Eye and Ear Infirmary during the afternoon, and was inclined to believe that the aural lesion was not responsible for the high temperature of the morning. At the time of my visit (4 p. m.) the temperature stood at 102.3° F. At midnight it rose to 107.2° F., a reading which was verified by Dr. Bowen, the

house physician, by the employment of a second thermometer. On the 28th the following temperatures were recorded: at noon, 99.3° F.; at 2 p. m., 102.4° F.; and at 4 p. m., 105.4° F. The last rise was preceded by a chill. Staphylococcus was found on examination of the smear.

The blood count was as follows:

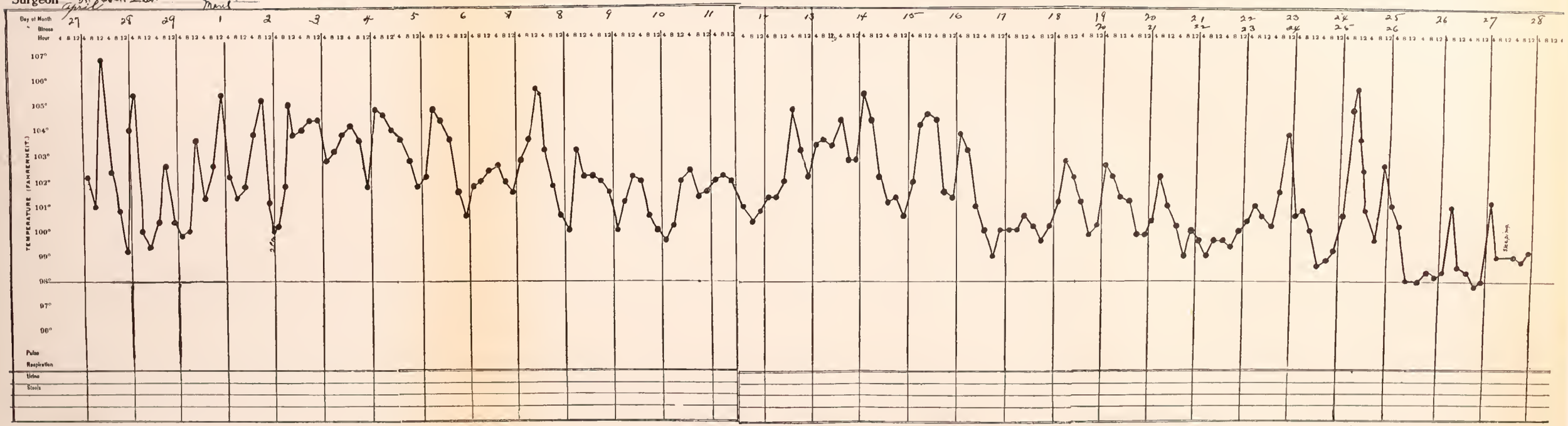
Red blood cells	4,640,000
White blood cells	18.800
Small lymphocytes	10.6
Large lymphocytes	1.
Transitionals	1.6
Polymorphonuclear	86.4
Eosins	.2
Mast	.2

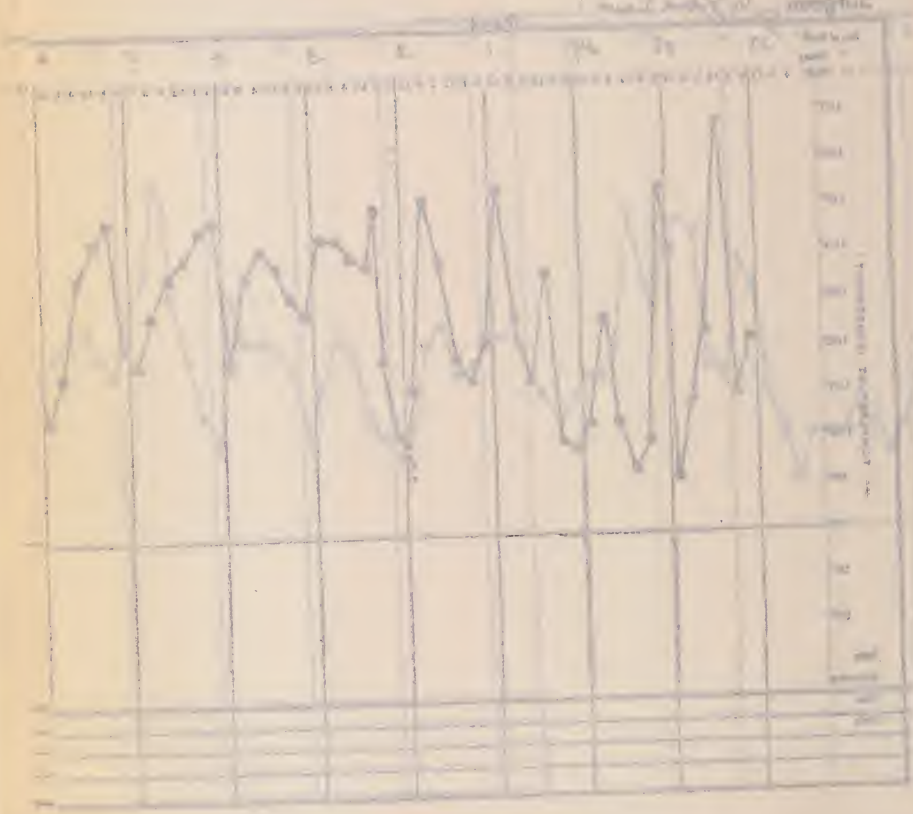
There were none of the physical signs of a mastoiditis. Dr. Henna could find no cause for the elevated temperature in any of the other organs of the body. It seemed therefore necessary to assume that a focus of infection must exist in the vicinity of the right ear: a diagnosis of septic thrombosis of the sigmoid sinus was made.

The seat of operation having been duly prepared the left mastoid process was opened; and upon the removal of the cortex and the superficial cells, the deeper-lying cells and the inner plate were found to be eroded, exposing the sinus throughout more than an inch of its length. The membraneous wall of the sinus was thickened, bathed in pus, and collapsed. After the mastoid cell structure had been thoroughly removed, the internal jugular vein was exposed and ligated at two points, and the intervening portion of the vessel excised. The facial branch of the vein was found to be almost as large as the jugular vein itself. After the removal of the excised jugular vein the sinus was opened, and it was found that the lumen of the vessel between the clot at the knee and the clot in the bulb was practically obliterated. Unfortunately, the vein and clot were lost in the laboratory, and I am therefore unable to state whether the infection of the vein and clot was the same as that found in the aural discharge, or not.

Name Baby J—

Surgeon Dr. Robert Lewis





For eight days following the operation, the infant showed all the evidences of a marked sepsis, as the accompanying temperature chart shows. On six different occasions the temperature rose to between 105° F. and 106° F. The elevation of temperature was usually followed by a decided fall, but no chills were observed.

The blood count on March 4th showed the following results:

Polymorphonuclear cells reduced to	59.2
Small lymphocytes	28.
Large lymphocytes	8.
Transitionals	4.

On the fourteenth, fifteenth and sixteenth days following the operation the temperature rose to between 105° F. and 106° F.; on the twenty-fourth day it reached 104.2° F.; and on the twenty-fifth day 106° F. Four days later still (twenty-ninth day following the operation), the child was discharged from the hospital, with the wound in the neck healed and the mastoid wound granulating well.

Four weeks later the mastoid wound had also entirely healed.

The interesting points in the case are the following:—the occurrence of extensive disorganization of the mastoid process, with involvement and collapse of the sigmoid sinus, but without any physical signs aside from a slight mucoid discharge from an ear the drum membrane of which was only a little congested and oedematous; the infective agent at the bottom of all these disturbances being found, on two separate examinations, to be only a staphylococcus.

Discussion.

Dr. Welty, of San Francisco. I simply wish to report two very interesting cases of sinus thrombosis that were not accompanied by fever or chill. The first case, from the country: acute otitis of four months duration with intense pain back of the mastoid, so intense was compelled to take morphine for the past four weeks.

The entire mastoid a necrotic mass. In wiping this away with sponges, an abscess cavity was opened; on more careful investigation it was found to be the sinus, which was curretted from either end until free bleeding. Jugular was not ligated because there was no general infection. Made uninterruptd recovery.

The second case, acute otitis of four weeks duration, last two weeks intense pain over the whole side of the head Patient looked very ill: no temperature or chill. Operated the same day. Large necrotic mastoid, sinus partially uncovered by disease, leaving a dark colored spot about the size of a lead pencil. The sinus was uncovered for an inch and a half; incised, clot removed and curretted from either end until free bleeding and packed. The jugular was not ligated for the same reason as stated before. Uninterrupted recovery.

These two cases are interesting because there are only some fifteen or twenty cases so reported. However, I am convinced that there are many cases that go unrecognized. Some years ago the autopsy surgeon of San Francisco found a few such cases. This latter is from hearsay information.

Dr. Frederick L. Jack, of Boston, described an incision for ligating the jugular which had been proposed to him by Dr. Tobey, one of his assistants. Instead of making the usual incision, parallel with the border of the sterno-cleido-mastoid muscle, one was made directly across this muscle, following the line of a small wrinkle in the neck. After making the incision to the platysma, the fibres of this muscle were separated at right angles to the incision in the neck. This procedure gave ample room for ligating the vein in the usual way. The resulting scar was hardly noticeable.

Dr. John B. Rae, of New York City, deprecates the practice of curretting towards the bulb before ligation of the jugular.

HEMANGIOENDOTHELIOMA OF THE TEMPORAL BONE.

By FREDERICK L. JACK, M D., Boston Mass.

Pathological Report By CALVIN B. FAUNCE, JR., M. D., Boston, Mass.

M. P., Armenian, 46 years old, entered the Massachusetts Charitable Eye and Ear Infirmary, on December 24th, 1911.

He had suffered considerable pain and loss of hearing before applying to the Infirmary for relief, and gave the history of a chronic suppurative inflammation involving the right middle ear and the removal of a polypoid growth about eighteen years ago.

Examination revealed a purulent discharge and growth, filling the middle ear cavity. Profuse hemorrhage followed the removal of a portion of the growth, for microscopic examination. The pain increased and at his next appearance two days later, he was admitted for operation.

His condition changed slightly. Some redness and swelling of the canal wall, with preauricular tenderness was noticed. No mastoid tenderness.

Microscopic Report: Rare tumor, exact nature undeterminable, because of insufficient amount of material.

On December 29th, the radical operation was performed. The bony cortex was found extremely thick and hard. Deep situation of the antrum and much inflamed. Nothing unusual was encountered, until the middle ear cavity was reached. This was found filled with a growth extending inward and upward, nearly three-quarters of the distance to the median line in the petrous bone. Attempts at the removal were followed by such profuse and uncontrollable bleeding and facial twitching, that further operative interference seemed inadvisable. The radical cavity was treated in the usual way.

Patient comfortable for two days when the pain returned in the ear, together with headache and rising temperature.

Leucocytosis 14,400. From that date his condition grew increasingly worse. Three days later white blood count was 19,800. Lumbar puncture showed turbid spinal fluid growth of fine streptococci. Death occurred on January 7th, 1912, nine days after the operation.

*Pathological Report.**

Autopsy January 8, 1912, at 11 a. m. Head only. Permission for general autopsy not obtainable.

Body is that of a well nourished man, cold, no rigor. Pupils equal 5 m. m. in diameter.

There is an open operative wound in the right mastoid region 6 m. m. in length. There has been an exenteration of the right mastoid bone. The bone cavity is bathed in white, creamy pus. Apparent exuberant blueish-red granulations protrude from the internal portion of the cavity and from the middle ear.

On removal of the calvarium the pia is seen to be congested. The sulci are everywhere oedematous and markedly purulent. There is no hemorrhage. The cerebro spinal fluid is purulent. The base of brain is covered with fibrino purulent exudate. The venous sinuses are free. Roof of tympanum is intact; there is no evidence of extension of the purulent inflammation through the petrous bone or the dura.

Projecting from the posterior surface of the petrous bone is a nodular tumor mass, 19½ m. m. by 19½ m. m. The tumor projects 8 m. m. beyond the inner table, replacing the bone and the dura at this point. The upper margin of the tumor extends over the internal auditory meatus, but does not reach the tentorium. The nodular surface of the tumor is smooth and is reddened and vascularized. It is nowhere adherent to the cerebellum, the opposing surface of which is normal, but for the meningitis.

On sectioning the specimen after decalcification the involvement of the bone is much more extensive than the projecting portion of the tumor seemed to indicate. The

*From Pathological Laboratory of Massachusetts Charitable Eye and Ear Infirmary.

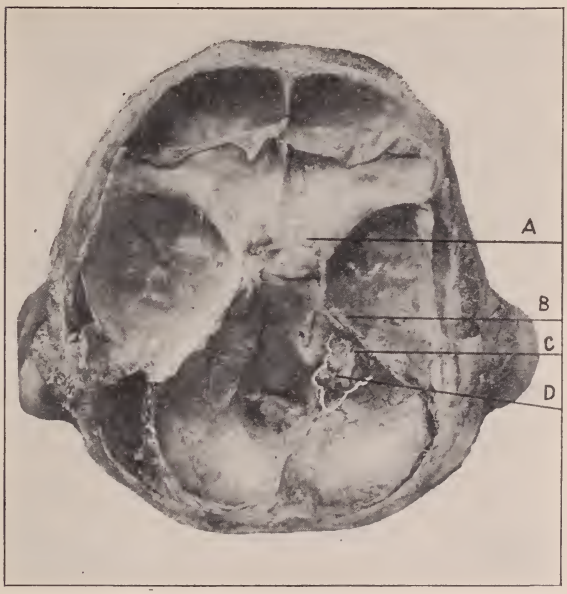


Fig. 1. A. Second nerve. B. Fifth nerve. C. Seventh-eighth nerves. D. Tumor.
Hemangioendothelioma of petrous bone (outlined in white) presenting in posterior fossa. The upper margin of the tumor involves the internal auditory meatus.

Fig. 2. Hemangioendothelioma of Temporal Bone (outlined in black). Showing destruction and invasion of the bone by the tumor.

entire petrous bone is pervaded by the growth including the labyrinth. The margin of the tumor is fairly sharply defined (Fig. 2). The tumor passes entirely through the temporal bone meeting the soft tissues beneath.

Smears taken from the purulent exudate show streptococci pyogenes.

The whole of the temporal bone including the tumor and part of sphenoid bone was removed, intact.

One-half of the presenting portion of the tumor was removed and fixed in Zenker's fluid. The remaining part of the tumor and the bone being fixed in Orth's fluid (Miller's fluid plus formalin), to which was added glacial acetic acid to the amount of three per cent. The bone was placed in fresh solution daily for one month. It was then washed in running water for 24 hours and decalcified in a solution of 6% nitric acid in 10% formalin. Some of the tissue was further decalcified in a saturated solution of picric acid. When thoroughly decalcified, the specimen was dehydrated in alcohol and imbedded in celloidin.

The structure of the tumor is best seen in the Zenker fixed specimen and is strikingly uniform throughout.

The internal surface of the tumor is covered with fibrin and pus; the superficial vessels show numerous purulent thrombi. The infection cannot be traced by direct continuity through the tumor, but that the infection undoubtedly took this course is shown by the infected air cells and medulary spaces passing from the site of the operation and reaching to within a short distance of the inner surface of the growth.

The tumor is made up of actively proliferating small and large vessels, most of which are somewhat larger than capillaries. As a rule the vessels have a very definite connective tissue wall and are lined for the most part by a single layer of endothelial cells. These cells differ from the normal endothelial lining of the vessels, in that they are very large and irregular, and form thick, uneven layers. Certain of the vessels possess more than one layer of these irregular cells. Free, large endothelial cells, many show-

Fig. 3. A. Portion of tumor presenting in cranial cavity. B. Site of operation.
Hemangioendothelioma of temporal bone extending from site of operation into the cranial cavity.

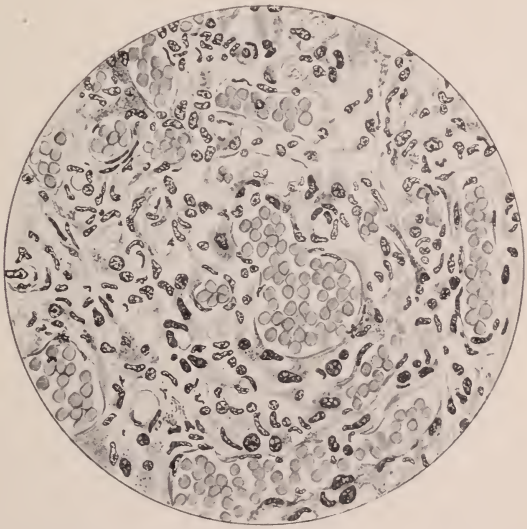


Fig. 4. Hemangioendothelioma of the Temporal Bone, showing proliferating vessels and endothelial cells.

ing signs of degeneration, are found between the vessels.

At the advancing margin of the tumor, the tumor cells first appear immediately around the vessels, where they gradually increase in numbers, replace all the tissue in the medulla, and eventually destroy and replace the bone.

The brain was hardened in formalin for one month and then carefully sectioned. No evidence of metastises were found.

Pathological diagnosis:

Hemangioendothelioma involving the temporal bone, including the middle and internal ear.

Chronic suppuration of the middle ear.

Operation for exenteration of right mastoid bone.

Streptococcus meningitis by extension through the tumor.

A CASE OF EXTENSIVE CEREBRAL ABSCESS, OF OTITIC ORIGIN, EVACUATING ITSELF THROUGH THE TYMPANUM.

By HENRY O. REIK, M. D., Baltimore, Md.

Mr. President, and Gentlemen, I might apologize to this body of distinguished otologists for presenting simply a clinical history, but that there are several interesting features in the case which, it seemed to me, would justify my placing it on record in this way. In the first place it represents a very striking example of one of the dangerous complications of neglected suppurative otitis media; a fatal brain abscess, developing insidiously, and killing quickly, after many years of an apparently harmless otorrhoea. Secondly, an unusual thing happened, in the spontaneous rupture of the abscess and its partial evacuation through the tympanum and external auditory canal. In the third place, the autopsy findings disclosed a very extensive abscess cavity that must have been in process of formation for a long time in its progress towards a vital spot.

I was called to the Baltimore Eye, Ear and Throat Hospital on the 23rd of June, 1911, to see a patient who had been brought in from a small town in West Virginia, and who was believed to be suffering from mastoiditis. The resident physician had, however, recognized the fact that it was not an uncomplicated condition. The patient was a man, 54 years of age, who said he had never suffered any serious illness, and who now complained only of severe and constant headache. On inquiry it was learned, in part from a friend who accompanied him, that he had been the subject of a chronic discharge from the right ear since early childhood, but he seemed not to have suffered any inconvenience or discomfort from this until within the last month. In the early part of June he begun to have marked headaches, diffuse in character and almost constantly present. At the same time the discharge

from the ear increased in amount. The pain in the head steadily grew worse, and for a week or ten days had been very troublesome. He stated that during the journey to Baltimore there had been at one time a sudden burst of pus from the ear and that the discharge had ever since been a constant flow; the hospital physician and nurse both noted the profuse flow of thick pus during the first few hours after his admission.

Examination showed a very marked tenderness over the right mastoid; he could not bear more than 200 grams pressure over the antral region. The temperature was sub-normal (97.6° F.) and the pulse high (90) and weak. He had been nauseated and vomited, at least once, on the train the night before. He had not been able to sleep, because of pain, but did not localize the pain, and neither pressure nor percussion discovered any sensitive areas except over the mastoid. The pupils responded to light and the eye-grounds were normal. There was no evident disturbance of muscle function, all the reflexes were normal, but, mentally, he was decidedly dull and apathetic. He answered questions intelligently but was disinclined to talk, and some of the delayed responses appeared to be due to slow cerebration.

Bacteriological examination of the pus proved it to be of unmixed streptococcus origin. The blood count gave leucocytosis of 16,500; with poly-morphonuclears of 92% and eosinophiles of only .2%.

The condition was diagnosed as mastoiditis with, probably, cerebral abscess in the temporo-sphenoidal lobe, and the patient was ordered to be prepared for operation at an early hour the following morning. During the night no new symptoms developed, the temperature did not rise above 99° F., but the pain in the head abated somewhat.

Operation disclosed the fact that the mastoid process was a mere shell of bone filled with pus, all the cellular structure here and the contents of the tympanum having been destroyed by the long standing purulent process. The cortex, which was very dense, was opened by aid of the Doyen's drill, and the necrotic masses in the mastoid removed with but little effort

by curettage; no chiseling was necessary. The bony roof of antrum and tympanum had been destroyed by necrosis and there was clearly visible a large perforation in the dura at this point. After thorough cleansing of the mastoid cavity and tympanum a more careful examination of the dural perforation was possible, and it was found to have been produced by rupture of a tempero-sphenoidal abscess. The necrotic opening in the bone was nearly circular and about 6 mm. in diameter, and that in the dura was nearly as large. The dura was firmly adherent to the healthy bone around the entire circumference of this perforation. A few drops of pus oozed out of the dural opening, and on introducing a graduated probe it seemed to drop into an abscess cavity, the bottom of which was 70 mm. from the suprameatal spine; which meant approximately 50 mm. within the brain substance. A small canula was then inserted and the abscess cavity gently washed out with normal saline solution, but only a small amount of pus and detritus was removed.

Feeling that we had probably secured satisfactory drainage for an abscess that had practically emptied itself, and there being no evidence to justify further exploration, it was decided to insert a small cigarette gauze drain and to await further developments.

For the next three days the patient was fairly comfortable, his temperature ranged from 99° F. to 100° F., only once touching 101° F., and then only for about two hours, but the pulse continued high. The wound was dressed and found in good condition, practically no free pus but the gauze drain was saturated. Irrigation with normal saline solution and the insertion of a fresh drain was all that was attempted at this dressing. On the following day temperature and pulse dropped to nearly normal and the patient seemed very much improved. This satisfactory condition continued for three days and we were feeling quite hopeful of his recovery when, quite suddenly the violent headache returned, the temperature jumped quickly to 103° F., accompanied by a weak pulse, rapidly rising to 140, and a state of semiconsciousness.

A second operation was immediately performed, consisting of an exploration through the temporal region in the usual site. The dura on the lateral surface was healthy looking, and a grooved director passed in various directions into the brain substance failed to find anything abnormal until it was passed directly inwards in a horizontal position, when it entered the same abscess cavity we had already explored from below. The patient's condition grew rapidly worse, he never regained consciousness, temperature rose steadily to 106° F. and the pulse to 160 in the course of the next 36 hours, at the end of which time he died

The accompanying illustrations show very clearly the autopsy findings, which were as follows:

Except at the points of injury on the under and lateral surfaces of the tempero-sphenoidal lobe, the external appearance of the brain and meninges was normal. On removing a section of the lateral surface, 15 mm. in thickness at its centre, a large abscess area was exposed, the depth of which was 50 mm. measured in the direction inward, upward, and forward from the opening over the tegmun antri; depicted in Fig. 2. A second section of 15 mm. thickness was removed and shows the inner wall of the abscess cavity as seen in Fig. 3. Here it will also be noted that there is a considerable area of inflamed white substance above and in front of the abscess wall. In fact, the abscess itself extended upward and forward almost to the sylvian fissure and internally to a depth of 55 mm. from the external surface; the total thickness of this hemisphere at the central point was 85 mm. The area of inflammation was clearly visible to the naked eye to a point a little above and anterior to the lateral ventricle. The sudden fatal termination was probably due to pressure in this region.

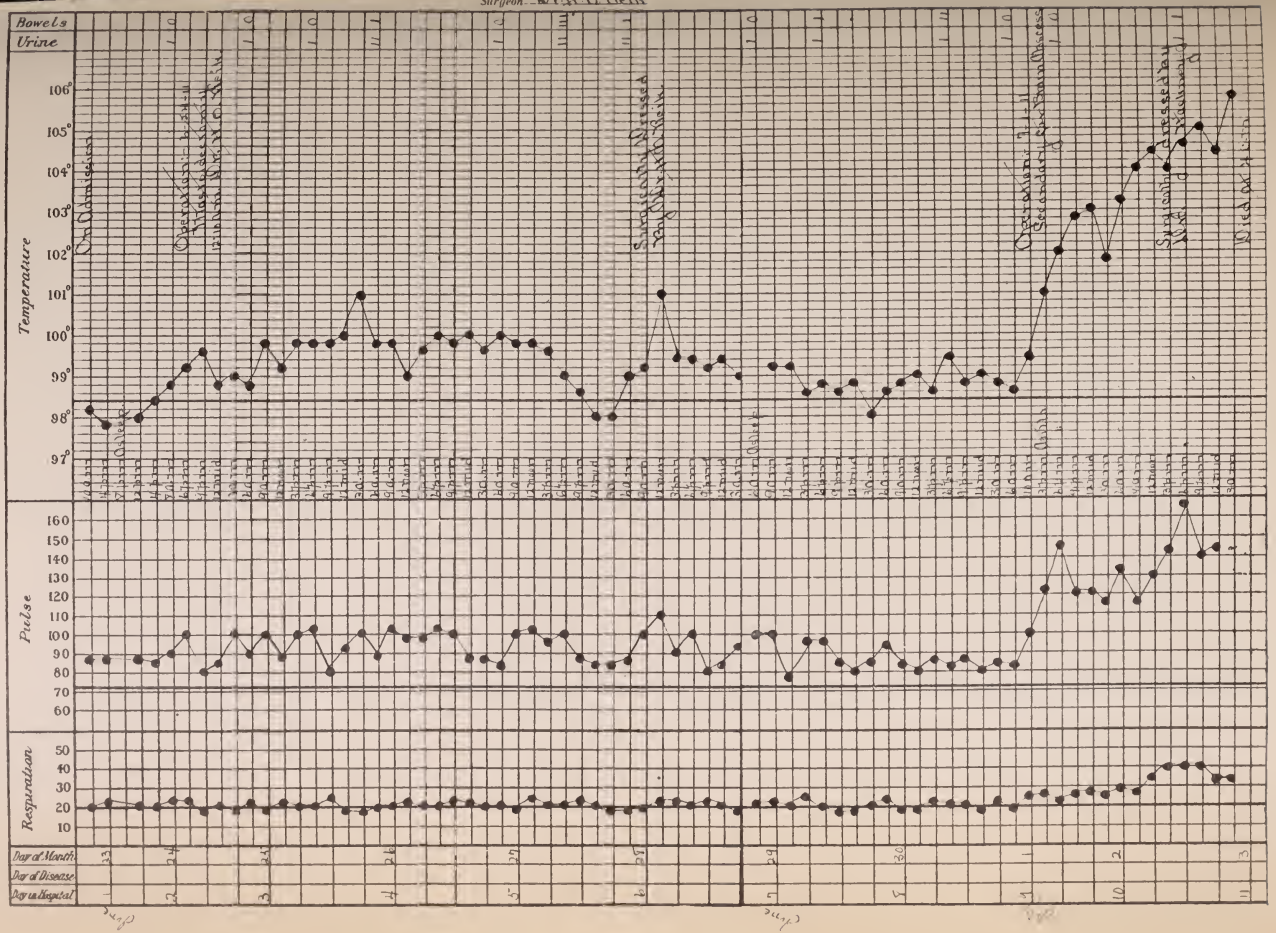

Surgeon
Bowels
Urine
Temperature
Pulse
Respiration
Day of Month
Day of Disease
Day in Hospital
On Admission
Surgically Dressed by Mr. H. O. Reik
Operation 7-11 Secondary Cyst Brain Abscess
Surgically dressed by Mr. H. O. Reik
Died at 4 a.m.

Fig. 1. Showing base of the right hemisphere. a. Marks the site of cerebral invasion and point at which the abscess ruptured into the tympanum. b. Marks the point of the exploration on the lateral surface of the temporo-sphenoidal lobe.

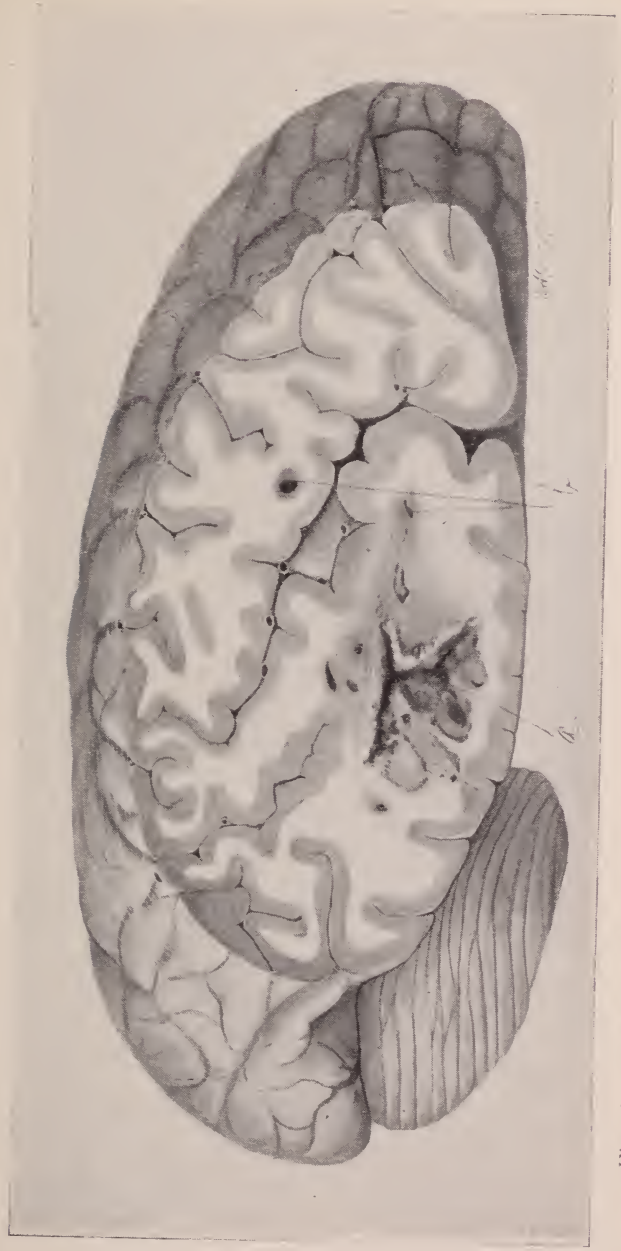


Fig. 2. Lateral surface of right hemisphere with a section 15 mm. in thickness has been removed, exposing the abscess cavity.

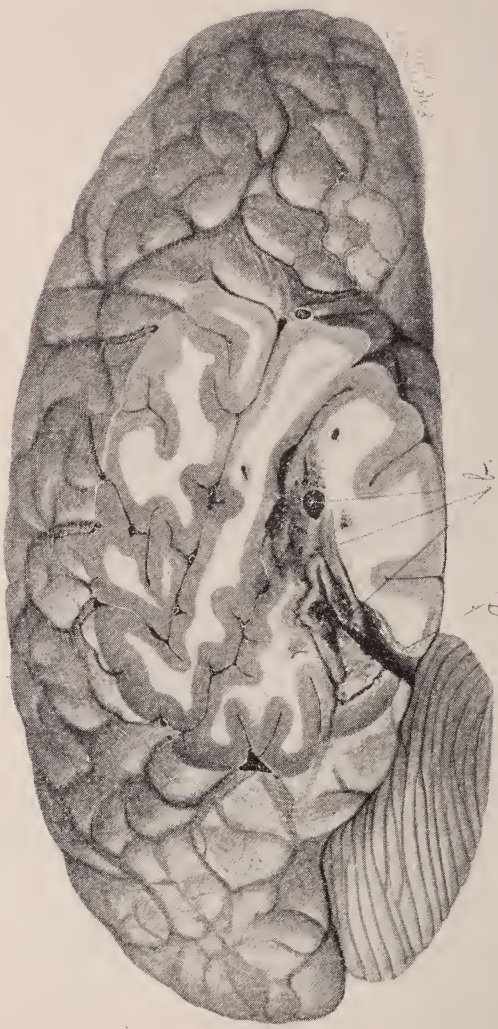


Fig. 3. a. Showing abscess cavity at a point 30 mm. from the external surface of temporo-sphenoidal lobe. b. Areas of inflammation beyond the abscess.

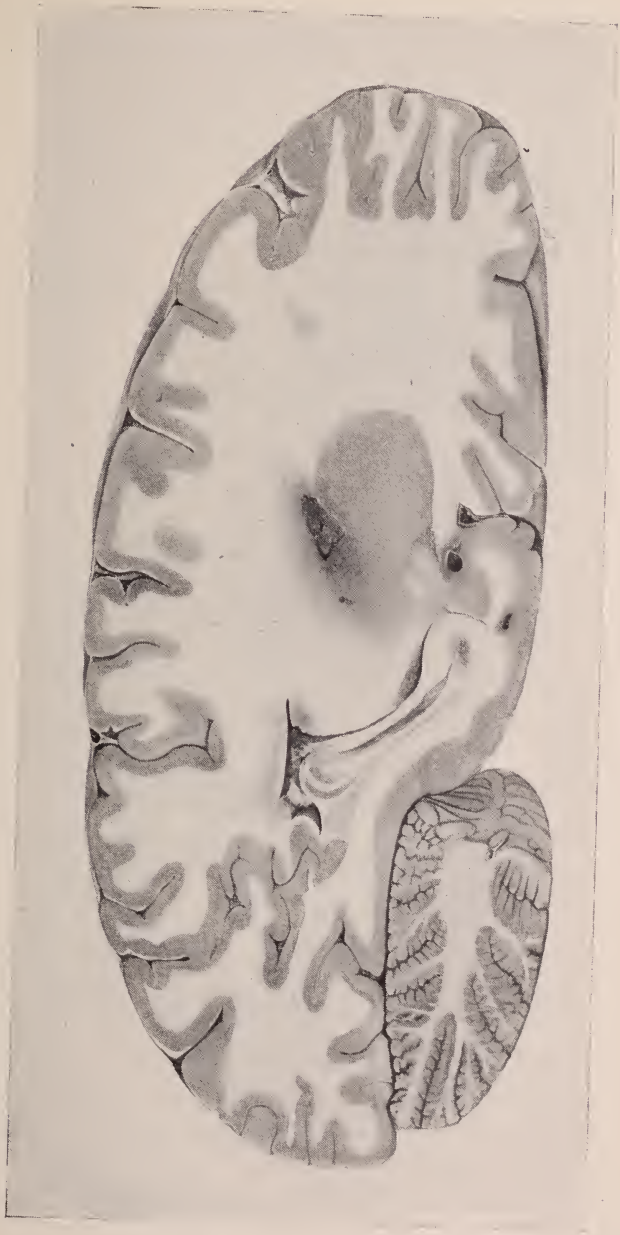


Fig. 4. Section made at depth of 45 mm. from surface.

"HOW MUCH TRAUMATISM WILL THE FACIAL NERVE WITHSTAND WITHOUT SACRIFICE OF FUNCTION?"

By DR. JOHN F. BARNHILL, Indianapolis, Ind.

Modern treatment of the suppurative diseases of the temporal bone has led the aural surgeon into greater and greater depths of this complicated structure until at present scarcely any part escapes interference when invaded by infective processes.

This extensive surgical invasion of the ear and its osseous environment may necessitate a considerable exposure of the facial nerve in its passage through the temporal bone. The pneumatic structure of this bone is such that in individual cases it occupies a deeper plane than the fallopian canal. When, therefore, these deep air spaces are invaded by infection it becomes necessary for the surgeon to remove the diseased structures not only down to the nerve but even beyond the nerve itself, thus exposing the latter to such manipulation as may be necessary to carry out correct surgical principles. In the surgery of the labyrinth the facial nerve must sometimes be exposed and handled, and in dealing with a thrombosis of the jugular bulb the trunk of this nerve must usually be uncovered, handled to a greater or less extent, and must sometimes be relocated as to its course either temporarily or permanently. Again it is not uncommon in the performance of the radical mastoid operation to find that the seventh nerve must be dealt with, either because of accidental approach to the trunk, or because the disease for which the operation is performed is found to include the fallopian canal. Indeed, there is no case of mastoid surgery, however simple it may seen, that may not involve the necessity of dealing with this nerve as a part of the procedure. In view, therefore, of the occasional necessity which arises of handling this important structure, during surgical pro-

cedures in the temporal bone, and of the appalling results to the patient, and of the unfavorable criticism of the surgeon in case of serious injury to the same. I have carried on a series of experiments on the facial nerves of dogs to determine, in so far as possible in this way, how much handling and real injury the nerve would withstand without permanent impairment of function; and, when injury occurs how soon we may expect improvement or cure.

This work was carried out in the Dog Surgery of the Indiana University School of Medicine, covering the period from January to May, 1912, Prof. W. D. Gatch and Dr. Paul B. Coble assisting. All animals operated for this purpose were stock dogs from the kennels, and some of them had been operated one or more times for abdominal or other experimentation before the nerve operation was attempted. Several of the dogs were, at the same operation in which the nerve experiment was made, subjected to experimentation on the lateral sinus, the results of which were reported to the 1912 session of the American Laryngological, Rhinological and Otological Society. All operations were performed under the strictest aseptic precautions. The experiments here reported on the nerve represent only types of cases but show approximately an average result for all.

Dog 2, Feb. 6. An attempt was made to expose the facial nerve at its point of exit from the stylo-mastoid foramen, after which it was planned to chisel away as much bone as possible over the course of the nerve trunk through the bone. Owing to my incomplete knowledge of the position and environment of the nerve, and to the unusually dense structures in the dog, which lie about the point of exit, the nerve trunk was completely severed by accident before it was recognized. The two ends of the nerve were dissected free for a short distance and an end to end anastomosis was made with small silk sutures. Complete facial palsy was present when the animal recovered from the anesthetic a half hour later.

The subsequent course was as follows: March 21, no improvement. March 28, facial palsy remains complete.

April 8, no better. April 18, dog in perfect health, and is able to partially close palsied eye. May 11, still further improvement in facial movement. Dog killed and the nerve trunk dissected out by Prof. Burton D. Myers, who prepared the specimen for microscopic study, and furnishes the following report concerning the same:

"In dog No. 2, three months and five days intervened between the time of operation and the date when the animal was killed and the nerve removed.

The nerve was cut off at the mouth of the stylomastoid foramen, three millimeters proximal to the original place of severance of the nerve. It was dissected out distally for two centimeters.

On examination of the sections of the nerve, it was found that a very complete regeneration had taken place, as evidenced by the great number of medullated nerve fibres just distal to the lesion. These fibres, surrounded by a mass of scar tissue, are somewhat smaller than normal, due to the thinness of the medullary sheath.

Under low magnification the nerve has much the appearance of a non-medullated nerve, due to the presence of an abnormally large number of nuclei in the neurilemma."

Dog 11. Operation April 10. In this case the nerve was exposed by cutting down on it at the point of its exit from the fallopian canal. The trunk was exposed for three-quarters of an inch, was then pinched by means of dressing forceps, was lifted from its bed, using estimated ten pounds traction, and was otherwise roughly, but not forcibly handled. When the dog was thoroughly recovered from the anesthetic it was found that no facial palsy was present. Professor Gatch, Dr. Coble and myself were surprised at the amount of traumatism inflicted on the nerve without apparent impairment of function. April 17, perfect facial movement, dog perfectly well.

April 18, no facial palsy, wound perfectly healed. Dog killed in order to make post mortem examination of the result of certain abdominal operations previously performed. The condition of the operated nerve trunk was not exam-

ined for the reason that no external evidence indicated any injury to this structure.

Dog No. 9. Operated April 3rd. Left facial nerve exposed from point of exit at stylo-mastoid foramen to point of division into its several branches. The trunk was then completely freed from all surrounding structures and was lifted by means of a blunt hook into several new positions. The sheath of the nerve was divided and a milk culture of streptoicocci was inserted under the sheath by means of a platinum loop. Wound closed leaving drain in position.

April 5, no constitutional symptoms, no paralysis; wound looks well. April 8, no palsy, wound suppurating. May 17, wound completely healed; no impairment of facial movement.

"Dog No. 3. Operated March 13. The facial nerve was exposed and dissected free the whole distance from exit to the point of division into its several facial branches. Trunk of nerve was severed near exit. The proximal and distal ends were laid in apposition and the surrounding structures were sutured over them, the skin being finally closed over all without drainage.

March 28, the facial palsy continues complete. April 8, no improvement. April 18, dog in perfect health and can partially close the eye on operated side. May 11, some facial movement, closure of eye better but not perfect; dog was killed under ether, the nerve trunk was dissected out by Dr. Paul B. Coble and was given to Prof. Burton D. Myers for examination. Dr. Myers made the following report on the progress in the repair of nerve trunk:

"Dog No. 3 was operated March 13 and killed May 11, so that fifty-nine days intervened between the time of operation and the time of killing.

The nerve was picked up a few centimeters from the stylomastoid foramen and dissected back to the mouth of the stylomastoid foramen. It was, however, impossible to determine with certainty that the proximal and distal stumps of the nerve had been united by suture.

A few centimeters from the lesion, there are many medul-

lated nerve fibers of perfectly normal appearance. These are probably fibres of the fifth N. (trifacial) which join with those of the facial in their course to the skin. These medullated nerves in the facial, a few centimeters from the stylomastoid foramen, may, therefore, be looked upon as a contamination of the 7th by the 5th.

Near the primary lesion, however, no normal nerve fibres whatever are found. The entire nerve trunk is made up of embryonic fibres, (Howell & Huber, Journal of Physiology, Vol. 13, 1892, pp. 335 to 406), or band fibres as they are called by Bethe, (Allgemeine Anatomie and Physiologie des Nervensystems, Zwölftekapitel, p. 182).

Occasionally, however, band fibres are found in which an axiscylinder is present, or an "axialstrang" as it is called by Bethe, by which term he means an incomplete axiscylinder, one capable of conducting impulses, but not having a complete formation of fibrillae.

These findings are, therefore, entirely in accord with the findings of Howell and Huber, Bethe, and others in regard to regeneration of nerves after they have been cut."

Dog No. 6. March 21. Right facial nerve exposed. Upper and middle branches dissected from surrounding tissues and ligated with heavy catgut leaving ligature on for seven minutes, then removing entirely. Contractions of facial muscles followed electric stimulation to either the proximal or distal portion of the nerve with ligature drawn very tight. This phenomena was probably due to the electricity spreading through the moistened tissues. Dog recovered from anesthetic in thirty minutes when facial palsy of right side, with ectropic lower lid, was observed. March 28, no improvement of facial paralysis. April 8, dog sick: cannot close eye; pupil dull and eye ball inflamed. April 14, dog died. Autopsy, at which time the nerve was dissected out and examined microscopically. The constriction caused by the ligature was still clearly visible on the nerve trunk.

Dog No. 5. April 20. Small white fox terrier—two abdominal operations three weeks ago—dog poorly nourished.

Technique very careful. Seventh nerve exposed where it crosses below cartilaginous external meatus and the three branches, an exterior, a middle and a posterior were exposed. The trunk was found by farradic stimulation to supply all face muscles. The trunk was cut close to the point of exit from the skull and the two ends immediately united by suture with split silk through the sheath of the nerve. Wound closed carefully in layers with interrupted fine black silk sutures. March 21, complete paralysis anterior branch, i.e., failure to close eye. Tears collect in eye. March 28, facial palsy still present Wound healed per prima. Inspection April 8 and 18, showed palsy still complete. April 18, eye still paralyzed. May 20, face still palsied. August 1, some movement of eyelid apparent.

Dog No. 4. March 13. Exposed facial nerve from exit to beyond point of division into its several facial and nuchal branches. Stimulated each branch, and the trunk with electric current in order to make sure that the exposed nerve was the facial. The nerve trunk was then completely freed from surrounding tissues, was roughly handled by rolling between the fingers and by lifting up with tissue forceps. The soft tissues and skin were then sutured over the nerve. No external dressing applied. March 21, winks eye perfectly. Wound healed per prima. Examination March 28 and April 18 showed facial movements perfect. On the latter date, however, the wound was suppurating and broken down while the dog appeared ill and died the next day. The condition of the nerve trunk was not examined post mortem for the reason that all agreed no appreciable injury had been done by the experiment of rough handling.

Dog No. 7. March 27. Left facial nerve exposed, and the trunk separated from surrounding structures for 2 c. m.; on electric stimulation all facial muscles responded. Cut the nerve trunk and sutured with three split silk strands passed through peri-neurium. After suturing applied electric stimulation with following result: proximal end, faint response due to spreading of current; distal end quick response. Wound tightly closed. Complete facial palsy when

dog recovered from anesthetic. March 28, complete paralysis. April 8, facial palsy continues. April 18, very slight closure left eye lid, although all observers agree palsy is yet quite complete. May 20, much improvement in facial movement.

It has been my belief, that the facial nerve in man bears manipulation badly and that its function is easily impaired or destroyed by handling. In the experiments on dogs here reported the trunk of the nerve was repeatedly pulled, pinched and otherwise roughly handled without the least subsequent appearance of impaired function. In several instances the pulling and pinching to which the nerve was subjected was so severe that it was my belief that function would certainly be lost as a result, but in no instance did palsy follow. In every case, however, in which the nerve was crushed, as in the jaws of an artery forceps by clamping, and where ligature was applied even though for a few minutes, facial palsy resulted immediately, and was as complete and permanent as if the nerve had been severed. Indeed, one dog whose facial nerve had been ligated for several minutes seemed to recover motion more slowly than did dogs on which the nerve trunks were completely severed, but which were immediately approximated by suture.

It was seen also, in several instances, that the nerve resists the presence of suppuration to a surprising extent. Several of the wounds suppurated freely without in the least impairing the function of those nerves that had been dissected free, had been roughly handled, but had not been severed or crushed.

The result of my experiments in completely severing the nerve, and then immediately approximating the ends and suturing them, seems to confirm similar experiments of others in that the time required for even partial restoration of function is three or four months. The experiments also seem to demonstrate clearly the fact that even when immediate approximation of a severed nerve is made degeneration of the nerve fibers takes place, and that primary union of the functionating parts does not occur.

It is possible that some of these cases in which the nerve was freely dissected and roughly handled, and in which subsequent infection of the wound occurred without any evidence as yet of disturbance of function, may later develop a palsy as a result of deposit of new connective or fibrous tissue about the nerve trunk. A longer period of observation will, therefore, be necessary to determine the final damage that may be done to the facial nerve from such injury to its trunk as here recorded.

Discussion.

Dr. Emil Gruening, of New York City, thought a great deal could be learned if the human facial nerve could be handled as Dr. Barnhill had handled the canine facial nerve. He had never seen paralysis of this nerve from merely handling it. He had touched it and had handled it with hooks without resultant injury. Where he had had facial paralysis he did not know he had done anything to the nerve.

Dr. E. E. Holt, of Portland, Maine, referred to a case which he had about twenty-five years ago, in which necrosis of the temporal bone took place in connection with suppurative inflammation of the middle ear, and a portion of the bone containing the nerve came away. Eventually restoration of the functions of the facial nerve took place.

He referred also to a case reported by Dr. J. Osgroft Tansley, of New York, in which three-fourths of an inch of the temporal bone containing the facial nerve came away as a result of an acute suppurative inflammation of the middle ear. The paralysis of the facial nerve which took place in this case disappeared in a few months.

Dr. B. Alexander Randall, of Philadelphia, hoped that Dr. Barnhill would present this work at the International Congress. He recalled one of Bezold's cases of exfoliation of the entire facial canal with paralysis which finally recovered. He had seen cases in which paralysis followed the simple mastoid operation, due to excessive packing, with eventual recovery.

Dr. Barnhill was right in thinking the human facial particularly easily injured as it rests in its bony canal. He would like to hear the experience of other members with anastomosis of the facial nerve. He had not seen a single case of recovery of paralysis worthy of discussion after attaching it to the spinal or hypoglossal nerves, even after two years, by this method, whereas he had seen recovery with massage after four years.

Dr. Welty, of San Francisco, Cal., cited a case in which he made anastomosis of the facial with the hypoglossal nerves, without result after two months.

Dr. William K. Rogers, of Columbus, Ohio, asked if Dr. Barnhill meant by traumatism injury similar to injudicious curettement.

Dr. Ralph Butler, of Philadelphia, called attention to a case in which on removal of a very large cholesteatoma, an inch of the bare facial within it was abraded by the curette. The facial paralysis, for a time complete, eventually recovered.

Dr. James F. McKernon, of New York City, during the past five or six years had seen several unsuccessful attempts at restoration of the function of the facial nerve after it had been cut.

Dr. Barnhill, in closing the discussion, referring to the question of the difference between traumatism outside and inside the fallopian canal, as suggested by Dr. Randall, said he had thought of this during his experiments, but could not follow up the idea on the dog. The nerve in the dog could not be exposed in the canal for a long enough distance to do very much work. There was no doubt about the greater danger of injuring the nerve in the bony canal. It was easy to suture the nerve in the dog. If sutured even at once it would degenerate back to the first ganglion on the central side and to the periphery on the distal side. The musculo-spiral, for example, had been resected for an inch and a half and yet had regenerated. There was a great deal yet to be learned

about nerve anastomosis, and it was to be hoped that others may carry forward the work. Referring to Dr. Gruening's suggestion that the nerve of the dog is capable of rougher handling than that of the human, the speaker said each animal differed in this regard from every other, and that this difference must be taken into consideration in experimental work. He did not curette the nerve, as suggested by Dr. Rogers. He had not had time to do everything as yet. He allowed some of the wounds on the dogs to become septic, for the reason that he wanted to see what sepsis would do to the nerve. He had found that it could be subjected without harm to a great deal more injury than he had imagined.

DECOMPRESSION FOR THE RELIEF OF DISTURBANCES OF THE AUDITORY APPARATUS OF INTRACRANIAL ORIGIN: REPORT OF THREE CASES WITH A PREVIOUSLY UNDESCRIBED AURAL CONDITION.

By WELLS P. EAGLETON, Newark, N. J.

The beneficial influence of decompression for the relief of edema of the optic nerve with failing vision, from increased intra-cranial pressure, is now generally recognized.

The exact cause of the papilledema, its early appearance in some cases, and its long delayed or entire absence in others, is still not completely understood. The length of the optic nerve is favorable to the development of not only a neuritis from direct pressure, but also from an edema of the sheath itself. The latter is bilateral; the former is unilateral. Examination of patients and operative findings by Payton,* caused him to regard the late papilledema following brain tumor as due to general cerebral edema. But that there is some other factor than cerebral edema, or edema of the nerve sheath, is shown by the frequency in which one sided papilledema is seen in the early stages of brain tumors.

Cushing has demonstrated that many of the palsies, such as the third and sixth nerve palsies, occurring at a distance from the growth, are due to direct pressure from disturbance of the circulation. That papilledema does occur from direct pressure, I observed in a case of a large hemorrhagic pial cyst of the frontal region on which I operated, the cyst having caused a marked homilateral optic neuritis with hemorrhages, with but very slight involvement of the opposite.

All opthalmologists have observed patients suffering from marked optic neuritis without any appreciable diminution of the central vision; and this may continue for a long time. One

*Tr. Ophth. Soc., of United Kingdom, 1908.

case that I have personally observed, has had a duration of over five years without failure of vision, while periods of one or two years are far from infrequent.

That slight pressure, properly applied to unprotected nervous tracts, may cause temporary suspension of function without any general increase in the intra-cranial pressure, is well known. This was exemplified in a case of temporo-sphenoidal glial cyst, causing hemianopsia, on which I performed a decompression in two stages. Following the first stage before opening the dura, the hemianopsia totally disappeared, although this had been known to be present for several months; the amount of pressure thus relieved being necessarily very slight.

Unfortunately we have not a method whereby we can diagnose a neuritis of the acoustic nerve before great reduction in the hearing occurs, although repeated, careful, functional tests of the cochlear and vestibular apparatus and of the relation of the different functional tests to each other, will enable us to do this, with more and more assurance, in the future.

The dural sheath of the acoustic nerve, unlike the optic nerve sheath, is very short, but that this space is frequently the seat of purulent effusion in meningitis is a frequent post mortem observation.

The degree of increased intra-cranial pressure which, with or without the other unknown factors, so frequently produces intense papilledema with subsequent total blindness, is manifestly insufficient to cause a similar loss of function of both auditory nerves, as bilateral deafness in cerebellar tumors is very rare. But the acoustic nerves must be subject, in subtentorial growths, to considerable pressure, edema and possibly to neuritis; the same degree of pressure and edema perhaps, which in the optic nerves would produce an edema discoverable by the opthalmoscope, but without marked diminution of vision.

Accurate microscopical pathological examinations of the auditory nerve are still so seldom made that an assertion of the frequency of acoustic neuritis is impossible. Again we have

little knowledge of the resistance of the acoustic nerve to mechanical pressure. Neuritis from toxic processes is clinically well recognized, and Orr, Rows and Milligan* have shown embryologically that there are two points in the nerve especially vulnerable to such invasion.

While the deafness associated with intra-cranial tumor may not be due to a neuritis and edema, still it is conceivable that such edematous pressure being combined with other factors, such as slight direct pressure, might cause a suspension of the function of the acoustic nerve at one of its two vulnerable points, enough to produce deafness; the relief of the pressure restoring the hearing. (The first and third cases reported certainly suggest such a view.)

If these contentions are true, then decompression in the early stage should have as beneficial an influence on the aural disturbances as it has on the papilledema; although as far as I am aware this has not previously been undertaken.

CASE OF BRAIN TUMOR ASSOCIATED WITH TOTAL DEAFNESS OF BOTH EARS AND NON-EXCITABILITY OF BOTH VESTIBULAR APPARATUS.—DECOMPRESSION.—TEMPORARY RESTORATION OF HEARING IN ONE EAR, THE VESTIBULAR APPARATUS REMAINING NON-EXCITABLE.

FIRST CASE: Mrs. F. B., age 38, Newark City Hospital, Dr. C. C. Beling's service. Neurological examinations by Dr. Beling. *Previous history:* Cancer of the left breast, operated upon. *Symptoms:* Severe pain in head, dizziness, nausea, diplopia. Attacks of "queer feeling" in hands, more pronounced in left, extending up to back of head; compelled to lie down and close eyes during attacks. Suddenly, one week ago, paralysis of left side of face and transient paresis and of right arm and leg. *Mental condition*: Emotional; suicidal tendency from pain in head. Occasionally forgets what is doing and saying. Great restlessness. *Examination*: Left sided facial paralysis; protrudes tongue to left. Grip of left hand less than opposite. Right knee jerk absent, left sluggish. Right plantar reflex absent. Left Babinski. No ankle clonus. *Eyes*: double papilledema, pupils equal, react to light and accommodation. Right vision: 20/30; left vision: counts fingers at 3 feet. *Hearing*: very poor in left ear; greatly impaired in right ear.

Four months later, returned to hospital with intense pain in head, unable to stand unaided. Gait shuffling; staggering, usually towards

*Jour. of Anat. and Physiol., Vol. XLIV, p. 127.

right. Speech thick with some slurring. Double facial paralysis, complete of left, partial of right. Patellar reflexes absent. Babinski negative on left side; slight on right side. Eyes: left corneal anaesthesia; left pupil smaller than right; intense papilledema of both eyes. Left vision nil. Right counts fingers at few feet. *For last three weeks totally deaf in both ears.*

Operation Oct. 19, 1911*:* Double decompression over both sides of cerebellum; removal of bone from mastoid to mastoid and from above transverse sinus to margin of Foramen Magnum. Dura not opened.

Six weeks after operation, headache and vertigo entirely relieved. Gait shuffling but walks around ward. Complains of numbness of thumb and first two fingers of left hand. Left facial paralysis still complete. Right side of face, although unable to close eye, lower part works well. No corneal anaesthesia. No sensory disturbance of face. Reflexes of upper extremity present. Patellar reflexes can only be produced by reinforcement. Plantar reflexes very sluggish. No Babinski. Papilledema entirely disappeared but optic nerves atrophied. Right vision counts fingers at two feet. Left same. *Hearing:* right ear, three weeks after operation heard loud voices, now *ordinary conversational voice heard at distance of four feet.* Left ear entirely deaf. *Vestibular apparatus*: No spontaneous nystagmus. Peculiar horizontal oscillation of eye balls. Rather jerky action in moving eyes in different directions. *Rotation, galvanic and caloric* (cold) *tests,* the latter applied in both ears separately and combined, *fail to produce any nystagmus or to cause sensation of dizziness.*

Later history: After several months of freedom from general symptoms of increased intra-cranial pressure, during which patient exhibited a tendency to over jocularity, gradual return of headaches, anaethesia and later ulceration of left cornea, gradual reduction of vision and hearing; and now, eight months after operation, left eye totally blind; right perception of objects; *total deafness of both ears.*

The important fact in this case was, that with total deafness of both ears, the removal of pressure by decompression, was followed by the restoration of the hearing of one ear to conversational voice at four feet, although the caloric, galvanic and rotary tests still failed to reveal any excitability of the vestibular apparatus.

While a positive diagnosis of the site of the lesion is perhaps unwise, both Dr. Beling and myself are inclined to regard the patient as suffering from an infiltrating pontine cerebellar angle tumor with occasional hemorrhages into its substance. Hemorrhage would account for the sudden paralysis of the face with transient paresis of the opposite

arm and leg, although this might be due to a transient circulatory disturbance, such disturbances being, we are convinced, from autopsy findings in cases which had previously been examined clinically, far more frequent than is generally recognized.

CASE OF PROBABLE TUMOR OF THE ACOUSTIC NERVE SHEATH—DECOMPRESSION—TEMPORARY DISAPPEARANCE OF ALL RETRO-LABYRINTHINE VESTIBULAR SYMPTOMS.

SECOND CASE, G. M. Age 28. Newark City Hospital. Dr. C. C. Beling's service. Neurological examinations by Dr. Beling. *History:* No previous illness. First noticed that could not hear well with left ear and for past two years total deafness of left ear. For past two years occasional "shooting pains" in arms, legs and face. During past six months "pains" very frequent, especially of left side of face, flank and abdomen; constant tinnitus; severe headaches; vertigo; sensation of rotation towards left. At present, vertigo so severe that can not walk without assistance, always falls towards left. The pains in left ear, face, and abdomen have recently become cramp-like and almost constant. Has not eaten food for several days. Nausea but no vomiting. Is afraid to sleep as fears will die. Slight tinnitus in right ear.

Patient states that on turning head to right everything moves up and down.

Examination: Facial expression drawn and anxious, fine facial tremor, no facial paralysis; hyperaethesia of left side of face; taste of left side of tongue lost, does not recognize salt or sugar; right side normal. Cramp of left masseter on opening mouth widely. Tongue protruded in median line. Tremor of both legs and arms. Fine tremor of right upper extremity. Attempt to grasp object with left hand evokes awkward movements, resembling athetoid movements; on being handed a dynamometer makes peculiar movements beginning in the little finger and extending in a rhythmical manner through fingers, wrist and forearm. The motions resemble those normally performed by the fingers, wrist and forearm in screwing, with the fingers and ball of the thumb, from left to right, a round-headed valve, placed with its top looking downwards. Left arm, pronation and supination rapidly continued, produces retardation and awkwardness of left hand, (diadococinesis). Dynamometer: L. 85-94-78-106. R. 78-18-90-100. Reflexes: Knee jerks normal; abdominal reflexes normal; no Babinski; no ankle clonus; no Oppenheim. Eyes: Slight paresis of left external rectus; marked *spontaneous horizontal nystagmus* on looking towards left; very slight rotary and horizontal nystagmus on looking to right; both pupils react to light and accommodation; right pupil at times slightly eccentric, and larger than left (spasticity of left), left corneal sensibility markedly

less than right; both fundi normal. *Hearing:* Left, total deafness; right, hearing for conversational voice apparently normal. *Vestibular reactions:* Left ear caloric (cold) followed by marked nystagmus in all directions. *(Left vestibular apparatus is hyperaesthetic.)* During induced nystagmus still falls to left in all positions of head. Right ear: Caloric (cold) causes no alteration in spontaneous nystagmus *(Right vestibular apparatus not excitable to cold.)*

Operation April 29, 1911: Decompression over both sides of cerebellum.

Following operation marked and rapid improvement in all symptoms.

Four months after operation: No headache, no dizziness, walks and works perfectly well. Can stand with eyes closed and feet together but when does so has sensation of falling towards left. No diadococinesis or disturbance of co-ordination of left upper extremity. No corneal anaesthesia, very slight nystagmus on looking towards left, which latter also still causes some mental confusion from the paresis of the left sixth. Right pupil still slightly larger than left. Patient apparently hears ordinary voices with left ear at distance of two feet. (This may not have been the fact as patient was not tested with a noise apparatus.) Tinnitus in left ear still present but slight and entirely relieved by lying on left side. No tinnitus of right.

Nine months after operation: Gradual return of nystagmus, and diadococinesis of left arm, but no headache or vertigo.

Present condition, 15 months after operation: Diadococinesis, and inco-ordination of left upper extremity; if directed to point has an oscillatory horizontal movement and then suddenly shoots arm forward. Slight contraction of color field of left eye, no interlacing. Long, slow, horizontal nystagmus on looking toward left; short, quick rotary nystagmus on looking towards right. Rotation causes a momentary increase of nystagmus away from lesion and adds rotary element to horizontal nystagmus towards lesion. *Rotation causes no dizziness. Caloric* (cold) *reaction negative in both ears;* although it is possible that when applied to the right ear it may at times momentarily lessen the horizontal nystagmus towards the lesion and quicken the rotary nystagmus away from the lesion, (paradoxical reaction.) *Galvanic tests have have no influence whatever.*

The importance of this case lies in the undoubted influence of the decompression on the nystagmus. For several months the patient apparently heard with the affected ear, but I have so frequently been mistaken in this that not having properly tested him with the noise apparatus. I am unwilling to state such to be the fact.

Cases 1 and 2 exhibited at meeting of Eastern Section, American L., R. & O. Society, held in Newark, New Jersey, Jan. 20, 1912.

The patient is probably suffering from a tumor beginning in the auditory nerve sheath and gradually pressing on the cerebellum, as is shown by the early appearance of the deafness followed later by fifth, sixth, seventh and ninth nerve involvement and then typical diodococinesis and inco-ordination of the upper extremity of the same side. The latter being typical of cerebellar involvement, as there is no accompanying reflex disturbance, no Babinski and the dynamometer records practically equal strength in both arms.

A CASE OF INCREASED INTRA-CRANIAL PRESSURE OF UNKNOWN ORIGIN, PROBABLY TUMOR OF THE CEREBELLUM PRESENTING: VERTIGO—SENSATION OF PRESSURE ON THE TOP OF HEAD—CONTRACTED AND INTERLACING COLOR FIELDS—CEREBRO SPINAL FLUID UNDER PRESSURE, WITH WHICH WAS ASSOCIATED; PROGRESSIVE DEAFNESS ENDING IN TOTAL DEAFNESS IN LEFT AND BEGINNING DEAFNESS IN THE RIGHT EAR. DECOMPRESSION FOLLOWED BY ENTIRE RELIEF OF ALL GENERAL SYMPTOMS, WITH PARTIAL RESTORATION OF HEARING IN LEFT EAR AND COMPLETE RESTORATION IN RIGHT EAR.

THIRD CASE, J. J. H., Fireman. Patient of Dr. H. Hart, first seen September 19th, 1911. *History:* Tubercular family history. About three years previously had attacks of dizziness, slight in degree. During last few months "dizzy attacks" have become so severe that is unable to attend to duties; attacks come on chiefly after eating. Sudden movement of head in any direction causes "blur" before eyes, accompanied by momentary confusion; moving objects, cars, smoke, etc., cause same sensation. Constant pressure on top of head; it is not a pain, but a sensation of weight, more severe at times than at others. For past few months constant tinnitus with deafness of right ear. *Examination:* B.P-120 m. m. Urine negative. Temperature normal. Neurological examination negative (Dr. C. C. Beling). Positive Von Pirquet's tuberculin reaction. Suspicion of Stellwag's symptom in both eyes; looking directly at object causes a contraction of the levator palpebra superioris, showing a line of sclera above cornea. Vision normal. Fundi normal, but contraction of the color fields with "interlacing" of colors. Right ear: Whisper heard at 1 inch; voice at 2 feet. $C^{128}AC$ not heard, BC normal. $C^{512}AC$ not heard, BC reduced 2 seconds. $C^{2048}AC$ reduced 8 seconds. Left ear whisper heard 4 feet. Vestibular apparatus: right ear, caloric (cold) no reaction. Rotation towards left, nystagmus 24 seconds in duration. Rotation towards right, nystagmus 25 seconds duration. *Rotation in either direction produces but a momentary sensation of dizziness.*

February 12th, 1912, (five months later). In spite of treatment patient has become progressively deafer, at times hearing much better than at others. Right C^{128}AC not heard C^{1024}AC heard, but very much diminished. Koenigs rods heard up to 20,000 v. s. Closure of poor ear does not cause increase of sound of tuning fork held on vertex. Vestibular apparatus: Both ears, *caloric* (cold) *gives no reaction* or vertiginous sensation. Following double caloric tilting head forward and away from affected side makes patient dizzy with some nausea. After double caloric left pupil larger than right; both rather dilated, pupil apparently made larger by lying down.

February 20, 1912. Lumbar puncture performed, fluid under great tension, about 20 c. c. withdrawn. Examination of fluid by Dr. Harrison S. Martland: "Quality clear, no flocculi. Noguchi's globulin reaction and Kaplan's modification of the butyric acid reaction, no increase in globulin. Microscopic examination: Scanty number of lymphocytes no tubercle, no other bacteria. Conclusion, normal cerebro-spinal fluid." Following the lumbar puncture no relief of aural symptoms.

February 28th, 1912, (one week later). Right ear with noise apparatus, voice heard at one foot; C^{128}AC not heard. BC greatly reduced; C^{512}AC not heard, BC reduced 13 seconds; C^{2048}AC not heard. Left ear whisper six inches, voice five feet, C^{128} AC>BC, AC reduced 9 seconds, C^{2048}AC reduced 6 seconds. Vestibular apparatus: caloric test (cold) in either ear, no reaction. Caloric (double), nystagmus of momentary duration immediately after water stopped, probably comes from left vestibular apparatus, not associated with any vertiginous sensation.

March 23, 1912. *Total deafness of right ear.* Vestibular apparatus: *cold and galvănic tests fail to give reaction in either ear.*

April 20th, 1912. *Operation*:. Cerebellum over right side exposed, dura not opened.

June 6th, 1912. Since operation absolute relief of all vertiginous symptoms. No pressure sensation on top of head. No blur before eyes on movement of head. Patient regards himself as perfectly well. *Examination:* Color fields of both eyes which had repeatedly been taken and prior to operation always found greatly contracted, with "interlacing," now normal of left eye, slight contraction of right; no color interlacing. Right ear: with noise apparatus, whisper heard at one foot, voice three feet. C^{128}BC>AC, AC reduced 8 seconds. C^{512} BC >AC, BC reduced 6 seconds. C^{2048}AC reduced 4 seconds. Left ear: voice ten feet C^{128}AC>BC, AC reduced 2 seconds, C^{512}AC>BC, BC reduced 6 seconds. C^{2048}AC reduced 3 seconds. Vestibular apparatus: rotation towards left, nystagmus 24 seconds; caloric (cold) right ear, no nystagmus.

The three cases, taken together, demonstrate many points of differential diagnosis between labyrinthine and retro-labyrinthine lesions.

In G. M. the long continued, spontaneous, horizontal nystagmus towards the affected side suggested an intra-cranial origin. With this he invariably fell in the same direction, and this in all positions of the head. The diagnostic value of this absence of the normal relation between the direction of the falling, the direction of the nystagmus, and the position of the head, in intra-cranial lesions, has been fully demonstrated by Bárány.

F. B. presented "disassociation" of the vestibular and cochlear portions of the nerve. After the decompression, while the cochlear portion of the nerve was functionating, shown by her ability to hear, the vestibular apparatus was not excitable by any known method. The importance of "disassociation" in the diagnosis of retro-labyrinthine affections, was described by Whittmaack and Frey. While several cases of disassociation from toxic affections of the nerve have been reported, as far as I am aware no case has previously been recorded of "disassociation" having been caused by restoration of the cochlear functions alone, by operation.

Both G. M. and J. J. H. had the great dissimilarity between the degree of the vertigo complained of, and that produceable by artificial stimulation of the vestibular apparatus, to which Bárány has called attention. J. J. H. in particular, was subject to "dizzy attacks" severe enough to incapacitate him, but turning produced practically no dizziness.

Both G. M. and J. J. H. presented a condition, viz: reduced reactiability to cold and to the galvanic current of the vestibular apparatus of the apparently unaffected side, the presence of which, in intra-cranial lesions, has not, as far as I know, been previously described. This condition, if generally present, may be of service in diagnosing increased intra-cranial pressure involving the auditory apparatus, possibly at an early stage.

G. M., when first seen, had, I thought, a hyperaesthesia of the vestibular apparatus of the affected side, as cold water syringed into the affected ear had a marked effect; but even at this stage it was impossible to produce any reaction by cold water when applied to the unaffected ear. Later neither

cold or galvanic current produced any effect. In J. J. H. it was impossible at any stage to cause any reaction by either cold or the galvanic methods applied to either ear, although double caloric at times apparently had a momentary effect.

Both G. M. and J. J. H. reacted to turning during the entire course of the disease. In G. M. turning, in the early stages, produced a nystagmus of unusual strength and duration. Later, the nystagmus was very slight, exemplifying the early hyperaesthesia and later diminished excitability described by Neumann as occurring in different stages of intra-cranial lesions involving the vestibular apparatus. In J. J. H. turning in either direction produced nystagmus of rather short but equal duration.

Reduced excitability to cold and to the galvanic current, of the apparently unaffected side, associated with induced nystagmus of equal duration from turning, in an intra-cranial lesion, although apparently paradoxical, is logically to be expected from the known, secondary reactions occurring months after the total destruction of the peripheral organ, by disease. While the immediate effect of destruction of the labyrinth is to cause a great difference in the duration of the induced nystagmus to turning in different directions, months later, it is found that the duration has become equal. On this fact Ruttin diagnosed a very old, total destruction of one labyrinth. Operation confirmed this. In the same class of cases reduced excitability of the normal labyrinth to cold is recorded as occurring by Bárány. Reduced excitability of the unaffected side to cold, and equal duration of the induced nystagmus from turning, can then be regarded as manifestations of the end result of the central mechanism, in its re-establishment of the disturbed compensation following destruction of one peripheral organ. Logically, the same should be much earlier expected in a slowly growing lesion of one side of the central mechanism itself.

As with the vestibular apparatus, so also with the cochlear apparatus, careful examination in the case of J. J. H. revealed a beginning deafness of the apparently unaffected

side, although he made no complaint of such and apparently was unaware of its presence.

The study of these three cases leads me to believe that in intra-cranial lesions of the posterior fossa, there may occur early reductions of the excitability of the vestibular apparatus, not only of the affected but of the unaffected side and perhaps an oscillation of the function of the cochlear apparatus of the unaffected side; all due possibly to an edema of the nerve sheath, which conditions may be revealed by repeated, careful examinations by qualitative tests and can be relieved, at least temporarily, by decompression.

Discussion.

Dr. Welty, of San Francisco, said the diagnosis of cerebellar tumors had been rendered comparatively easy by the ear findings. The differential diagnosis between cerebellar and acousticus tumors had also been rendered easy by the ear findings. The acousticus tumors destroyed the auditory function—while with other forms of tumors there would be remnants of hearing left.

THE INSIDIOUS LATENT PERIOD OF STREPTOCOCCUS MUCOSUS.

By FRED WHITING, M. D., NEW YORK, N. Y.

The respect which otologists have long entertained for the infective properties of the numerous varieties of streptocci with which they have to deal, has not, with the passing of the years, been in any wise diminished, but, on the contrary, appears with the accumulated experience of wider observation to have been greatly enhanced. That one particular variety of streptococcus should disclose a malevolent propensity peculiar to itself and more virulent than others of its kindred, seems, in view of the unrivalled capabilities of the entire genus for causing trouble, quite unnecessary. But repeated unfortunate experiences during the past two years have impressed upon the writer the conviction that streptococcus mucosus possesses a most dangerous and insidious latent period which may justly occasion apprehension concerning the outcome of any inflammatory ear disease which has its origin in the activities of this infective agent.

An observation which I am sure many of us have made in cases of threatened mastoiditis due to streptococcus mucosus concerns the disposition upon the part of this germ to institute a period of latency or apparent loss of activity, during which all the more acute manifestations subside, the inflammation apparently abates, and the apprehensions of both patient and doctor are gradually allayed. After a lapse of time varying from a few days to as many weeks, we are at length induced to operate by the reappearance of severe acute symptoms, it may be, or because the patient, although not presenting entirely convincing evidences of mastoiditis does not satisfy us, does not appear to be doing just right. In other words, we are impelled to the step by that inexplicable motive which must

in the last analysis be denominated as surgical judgment. In any event, we often find at the operation that extensive destruction of the mastoid had taken place quite out of proportion to the moderate severity of the earlier inflammatory manifestations, thus clearly indicating that although all subjective and objective symptoms had been in abeyance, a destructive osteo-myelitis had been steadily progressing, had, in fact never been arrested at any time.

Should the patients recover, as they usually do, notwithstanding our belated ministrations, we may heartily congratulate ourselves upon having escaped such an embarrassing situation at a cost of no greater humiliation to our self-esteem than a shock to our diagnostic astuteness.

So much for the vagaries manifested by streptococcus mucosus previous to operation, and sufficiently troublesome they would be if they offered no further problems to perplex us, but my personal experience with this germ has within the last year been supplemented by observations of its capacity for making mischief, not only previous to operation but also long after operation has been done, and almost up to the time of complete healing of the wound, which seems to me to be worthy of being carefully noted and brought to the attention of otologists.

Under ordinary circumstances, when we operate for mastoiditis in any case in which the symptoms previous to operation are not of such character as to lead us to anticipate an intracranial inflammation, and when the operation does not reveal the presence of such a complication, we feel justified in assuring the relatives and friends of the patient that a favorable outcome may be anticipated; but in the light of recent experience with this virulent germ, I feel that the prognosis should be very guarded, not only at the time of operation, but even at a much later period.

Bearing upon and illustrating the question at issue are three cases the histories of which I desire briefly to present.

Case I. A young man, thirty years of age, who had never had any ear inflammation until one month before he con-

sulted me. He was attacked with grippe, and after a few days complained of pain in the right ear. He was attended by an ear surgeon, who did a myringotomy, and advised irrigation of the ear. The discharge continued for three weeks, when the patient, becoming dissatisfied with the progress of his case, sought other advice and was told that a mastoid operation was necessary. This advice, not commending itself to him, he applied to me, and was advised that an immediate operation was imperative. The patient entered the New York Eye and Ear Infirmary, and a culture taken from the ear discharge was found to contain streptococcus mucosus (capsulatus). His blood examination showed a total leucocyte count of 4,200, and a polymorphonuclear percentage of 80. Thinking that this very low total count might be due to an error in computation, I had another count made a few hours later, which again showed 4,200 leucocytes, and about the same polymorphonuclear percentage as before. I expressed to his family my concern at the very low count, as indicating lowered resistance, and operated upon him the next day, finding a very extensive osteomyelitis of the mastoid process, which had destroyed the inner table of the skull in the middle cranial fossa over an extent as large as a silver half dollar. The exposed dura was covered with fibrin and small granulations, but no necrotic or softened area of the meninges could be detected. The sigmoid groove presented a surface of dense white bone, and the sinus was not exposed. The patient had a somewhat slow convalescence, without any noteworthy phenomena, and left the hospital on the seventeenth day, with his middle ear entirely healed, and his hearing excellent. During his stay at the Infirmary, repeated blood examinations were made, and in no instance was the leucocyte count above 4,600. He came to my office for dressings. The wound granulated in the most gratifying manner, and was apparently healing rapidly and kindly, the patient was regaining his strength and vigor, and with the exception of occasional neuralgic pains in different parts of the head, registered no complaints. He had, in fact,

arranged to resume business within a few days, when, on May 8th, six weeks after his mastoid operation, he suddenly experienced a sharp chill, followed speedily by a rise in temperature to 104°. The family physician reported his illness to me by 'phone, and was requested to have a blood examination made and report to me later. The blood examination showed total leucocytes 18,000, polymorphonuclears 82%. He was seen by Dr. Libmann in consultation, who reported slight rigidity of the neck, and well marked Kernig, more pronounced on the right or corresponding side. A blood culture was taken by Dr. Libmann at this time, and was subsequently found to contain a few scattered colonies of streptococcus mucosus. This finding was corroborated by blood culture made the following day by Dr. Dixon, when two colonies only of streptococcus were found in 20 cubic centimetres of blood. The patient's hearing in the affected ear was excellent. There was no vertigo, nystagmus, or other manifestation referable to labyrinthine involvement.

On my advice, he was placed in an ambulance, brought to the hospital as quickly as possible, and immediately taken to the operating room. Lumbar puncture under general anaesthesia showed the cerebro spinal fluid to be under great pressure, for it spurted forcibly from the canula, and when caught in centrifuge tubes was very turbid in appearance and filled with flocculi. Upon immediate microscopic examination, this fluid was found to be full of pus cells, but bacteria were not at the time demonstrable. These, however, were found in abundance the next day upon the spreads made from the centrifugalized fluid, and were streptococcus mucosus—the original infecting agent. Upon examining the wound, I found it filled with firm, healthy appearing granulation, which when removed by curetting disclosed no softening or other indication of the source of the present infection. The lines of the original incision in the scalp were extended, and the flaps reflected sufficiently to permit a free exposure of the squama, which was rapidly removed by rongeur and a large area of dura laid bare. The dural vessels were engorged and distended, but no fibrin was

found upon the surface of the meninges. The tension was low, owing to the lumbar puncture already made. After flushing the wound, and rendering it as clean as possible, an incision was made in the dura directly over the situation of the antrum and one inch above the floor of the middle fossa. The soft coverings of the brain thus exposed were deeply injected. The tempero-sphenoidal lobe was aspirated in several directions, with a view to determining the possible existence of an abscess, with negative result. A rubber tissue drain was then introduced through the opening in the dura above described, into the subdural space, for the purpose of favoring escape of the cerebro-spinal fluid. Over the surface of the exposed dura, a sheet of fenestrated rubber tissue was laid, through a hole in the centre of which the end of the subdural drain protruded. The object of this rubber tissue sheet was to prevent the tegumentary flaps when readjusted from adhering to the dural surface beneath, and thus effectually interfering with the efficiency of the drain. The patient was placed in bed, ordered sufficient morphea sulph. to keep him comfortable, and urotropin grains 15 every four hours were prescribed, by the mouth, if the stomach would retain it, or by rectum if the stomach rejected it. The patient was more comfortable next day, with a slightly lower temperature range, but the gravity of the symptoms was not materially abated. On the following day he had more pain, and abducens paralysis of the left eye was noticeable. Rigidity of neck and Kernig, both sides, marked. On consultation with Dr. Libmann, lumbar puncture was again resorted to, and about 15 cc. of cerebrospinal fluid was withdrawn, which exhibited the same turbidity and flocculence as on the first lumbar puncture. The wound in the skull was examined and cerebrospinal fluid found oozing along the subdural drain, which was removed, replaced with a slightly larger one, and the patient returned to bed.

From this time on his condition grew gradually worse; stupor was succeeded by profound coma, the temperature rose to 107°, pulse to 180, and thirty-six hours later he died.

Autopsy was not permitted. We were anxious to obtain a post mortem examination, chiefly for the purpose of determining—in view of the finding of a few colonies of streptococcus mucosus in the blood—whether or not the sigmoid sinus was the seat of a thrombotic inflammation.

The wound of this patient, at the time of the chill and onset of meningitis, was apparently in a most satisfactory condition, and the granulations, which were firm and well nourished, had not been lacerated by curetting or crushed by the forcible introduction of gauze packing.

The absence of nystagmus and vertigo and the preservation of perfect hearing precluded the possibility that the labyrinth had provided the path of infection and emphasized the likelihood that the source of the trouble was to be found in lymphatic absorption.

Case II. The second case was that of a young man twenty-eight years of age, who came to the New York Eye and Ear Infirmary from one of the interior towns of the state. He gave a history of chronic suppuration of the left ear, following one of the exanthemata of childhood. Previous to the present attack he had never experienced any vertiginous or other manifestations referable to the labyrinth, and had for many years considered that the ear was deaf. How profound the deafness had been previous to entering the hospital, there was no means of determining.

Two weeks before he was admitted to the Infirmary he had noticed a cessation of the discharge from his ear, and coincident therewith the mastoid became sensitive. He suffered from dizziness, nausea, and vomiting, and was compelled to keep in bed, owing to the severity of the vertigo. No record of the temperature had been kept, and when admitted to the hospital he presented the following picture:

On physical examination he had slight horizontal and rotatory nystagmus toward the right, or opposite side, which was not influenced by the caloric test. His left mastoid was moderately tender to pressure over the situation of the antrum, but not elsewhere, and there was no soreness or

rigidity of the neck upon moving his head. Kernig's sign was absent, and upon ophthalmoscopic examination the fundus occuli was normal. No occulo-motor or other paralysis. The left ear presented a sufficiently ordinary picture of otitis media purulenta chronica. Very little membrana tympani remained except the annulus, and the only recognizable contents of the tympanum were the truncated remains of the malleus, which could be recognized protruding from a mass of granulations in the vertical meridian above. A scanty foul-smelling discharge was present in the tympanum, at several points upon the walls of which denuded bone could be felt by probing. Whether or not the stirrup was in position could with difficulty be determined, owing to the presence of a mass of granulations in the vicinity of the oval window, which bled sufficiently upon using the probe to obscure the field of exploration.

Upon functional examination, Middle C. Fork was heard only in the right or good ear, and when the buzzer was applied to the right ear there was complete deafness for all sounds in the left. The blood count showed total leucocytes 7,600; polymorphonuclears, 82%.

The patient was operated upon that afternoon by radical procedure, associated with complete ablation of the semicircular canals and cochlea. The semicircular canals, upon being exposed, were critically inspected for erosion or fistulous openings, but no such destructive process could be discovered, notwithstanding which reassuring appearance, the horizontal canal, when opened, contained granulations which extended into the vestibule. The contents of the posterior and superior canals, upon investigation, showed no macroscopic evidence of suppuration. After carefully removing the granulations about the oval window, no remnant or trace of the stirrup rewarded a conscientious search. Only an unobstructed opening was found through the crumbling walls of which a probe passed directly into the vestibule. The cochlear whorls were filled with granulations, and upon removing the bone (upper whorl,) at the apex of the modiolus

a single drop of thick creamy pus oozed out, which being at once transferred to a gelatine tube provided on the following day a specimen of streptococcus mucosus in pure culture. The inner wall of the cochlea appeared firm and smooth, and there was no fracture of the modiolus during operation.

Following operation, the patient's condition improved greatly, and in a few days his vertigo had so far subsided that he sat erect in bed without discomfort, was eating and sleeping well, and the date of his dismissal from the hospital and return home had been discussed. On the twelfth day after the operation, he complained of pain in his head, had a chill, and his temperature rose to 104°. He was taken to the operating room, and lumbar puncture revealed a turbid cerebro-spinal fluid, containing pus cells and streptococcus mucosus. The inner table of the skull in the middle and posterior cranial fossae was removed, dural flaps were made, and rubber tissue drains were introduced. There was no amelioration of the symptoms, and the patient speedily sank into a condition of coma and died two days later. Autopsy was urgently requested, but positively refused. The observation of chief interest in this case, as in the preceding one, will be found in the experience of an infection manifesting itself after a latent period of considerable duration, during which the patient, judged by the usual clinical signs, appeared to be making satisfactory progress toward recovery.

The after treatment had been conducted with great care, and no unnecessary probing about the labyrinth had been permitted. The outcome of this case would have been less keenly disappointing had the onset of meningitis followed promptly upon the operation, but when subsequent to operation the patient experienced marked relief, and for two weeks encouraged hopes of a speedy recovery, the ensuing rapid fatality was specially distressing.

Case III. One year ago, I was called to attend a lady aged eighty years, suffering with grippe. She complained of

pain in the left ear. When examined the drum was found bulging, and myringotomy was performed. A smear made from the discharge showed streptococcus mucosus (capsulatus) as the infective agent. The inflammation was not of severe character, and ran its course to healing in about three weeks, during which period the mastoid was never tender to pressure. A culture taken from the discharge a few days after the myringotomy demonstrated the presence of the same micro-organisms. Five weeks from the beginning of the attack the hearing was normal. This patient, whose advanced years precluded all violent effort or undue exertion, and whose affluent circumstances permitted her to surround herself with every hygienic precaution, enjoyed perfect health for the ensuing five months. At the end of that time, she complained of pain in the abdomen, a little to the right of the median line, and midway between the umbilicus and the symphysis pubis. Within a day or two an indurated area, very tender to the touch, was distinctly palpable at the seat of pain. The services of a surgeon were procured, and under an anaesthetic an incision was made in the abdominal wall, an abscess evacuated, the pus escaping from which was found upon culture to contain streptococcus mucosus. The wound healed kindly within a reasonable time. This patient had suffered no injury, bruise, or strain to the part affected by the inflammation, nor to any other part of her body, nor had she been troubled, so far as she knew, by sore throat or by any abrasion of the mucous membrane of the mouth or nose. Her temperature had been taken at least three times daily by her attendant during the last five or more years, and reference to this record shows that during the interval between the suppuration of the ear and the infection of the abdominal wall, she had shown no departure from normal. It seems a warrantable assumption, therefore, that no intercurrent inflammation had been operating which could reasonably be charged with originating the infection, which had resulted in abscess of the abdominal wall. The question then arises as to whether or not it is a legitimate conclusion that bacteria remaining in the

ear in a latent condition during these many months had suddenly, without local irritation, made their way by means of the circulation, to a site remote from the original focus, and there, finding conditions favorable for development, had regained their acute infective properties and caused an inflammation resulting in abscess.

1. The question for our consideration which these cases raise, and to which I desire to direct the attention of the members of this Society in thus briefly presenting them, is whether or not streptococcus mucosus, more than other varieties of streptocci, is prone to a protracted period of latency after operation, of which in the past we have not been accustomed to suspect it.

2. Has the existence of this latent period after operation been sufficiently well recognized in the experience of other surgeons to warrant an operator, in any case where the presence of this infection has been demonstrated, in giving a more guarded prognosis than would otherwise be justified?

3. Does not a moderately low polymorphonuclear count expose a patient having mastoiditis due to streptococcus mucosus to a grave danger, in addition to the usual operative risk?

4. Should not the knowledge that we are dealing with an infection of this nature (streptococcus mucosus) lead us to exercise *extreme caution in the after treatment of the wound,* lest by injudicious interference, such as unnecessary probing and rough manipulation of gauze, granulations may be lacerated and an absorptive surface presented which may offer a gratuitous path for serious and sometimes fatal infections.

Discussion.

Dr. Norval H. Pierce, of Chicago, had had a case in which the clinical course was very similar to that reported by Dr. Whiting, except that the staphylococcus, in pure culture, was obtained from the blood. The patient was a woman, twenty-eight years of age, who had had a discharging ear

since she was one and one-half years old. Cholesteatoma was found at operation. The blood count was between five and six thousand. Pus from the ear, obtained at the time of operation, showed pure culture of staphylococcus. The sigmoid sinus was exposed as was the dura of the middle fossa. On account of the microorganism present and also on account of the low leucocyte count, he neither opened the dura nor performed the radical mastoid operation. Everything went along beautifully for about ten days, when there was a rise in temperature, preceded by a chill. He opened the sigmoid sinus, but found nothing wrong there. He punctured the dura in the middle fossa, hoping that possibly he would find some pus beneath it. He found an adhesion between the dura and the pia. The woman died of chronic sepsis in the course of three weeks. Throughout the course of the trouble the only organism that could be found was the staphylococcus. This was identified by a well-trained bacteriologist. At no time was the leucocyte count above seven thousand. He had made a vaccine, which he tried out thoroughly, without the slightest influence.

Dr. James F. McKernon, of New York City, had had a similar experience. The mastoid operation was performed, the wound healed completely and the patient was going about. He then developed a temperature of 103-plus degrees F., with pain in the head, and chills, lapsing into a state of coma. He was operated upon, and the entire brain was found to be covered with pus containing the streptococcus mucosus. The speaker had always considered the streptococcus mucosus extremely virulent, and had never felt that his patients were free from possible infection for weeks after operation.

Dr. William K. Rogers, of Columbus, Ohio, asked whether, in cases of latent, delayed infection from the streptococcus capsulatus, antistreptococcic serum had been employed by others. He had never encountered any of these latent cases of infection, but he recalled three cases in which this form

of streptococcus was found primarily, one in a chronic case of mixed infection, and two in pure culture in acute cases. They were accompanied by a low leucocyte count and moderate temperature. Each of the three cases seemed likely to require mastoid drainage, but cleaned up through the natural passages, under local treatment, after the use of antistreptococcic serum. He wondered if the serum would not have had some influence on the case detailed. With the vaccines his experience had been the same as that of Dr. Pierce, but with the antistreptococcic serum he had had more success.

Dr. B. Alexander Randall, of Philadelphia, considered the antistreptococcic serum an antidote when a patient was already succumbing to the infection, but to give the bacterin under such circumstances was to add an additional burden to a patient about overcome. The antistreptococcic serum had done well in some cases of uterine infection, but it had to be given in tremendous doses to be of any value. He recalled some cases reported here twenty years ago. One, a woman had presented herself with a waning condition of middle ear inflammation. A month or two later this passed into an infection which he did not see, as it was treated by a general surgeon. The patient died of septic meningitis without one drop of pus from the ear. This showed that in the apparently very mildest form of the disease the most serious destruction might be going on.

A CASE OF OTOSCLEROSIS.

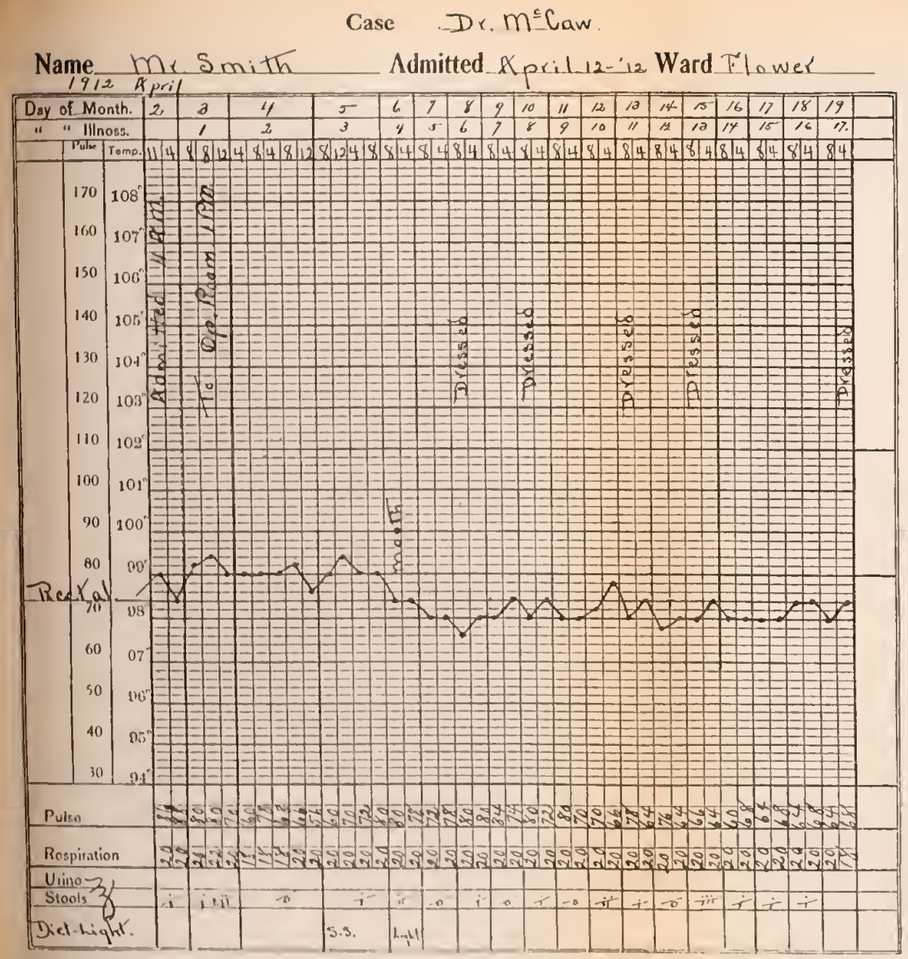
By W. SOHIER BRYANT, A. M., M. D., New York, N. Y.

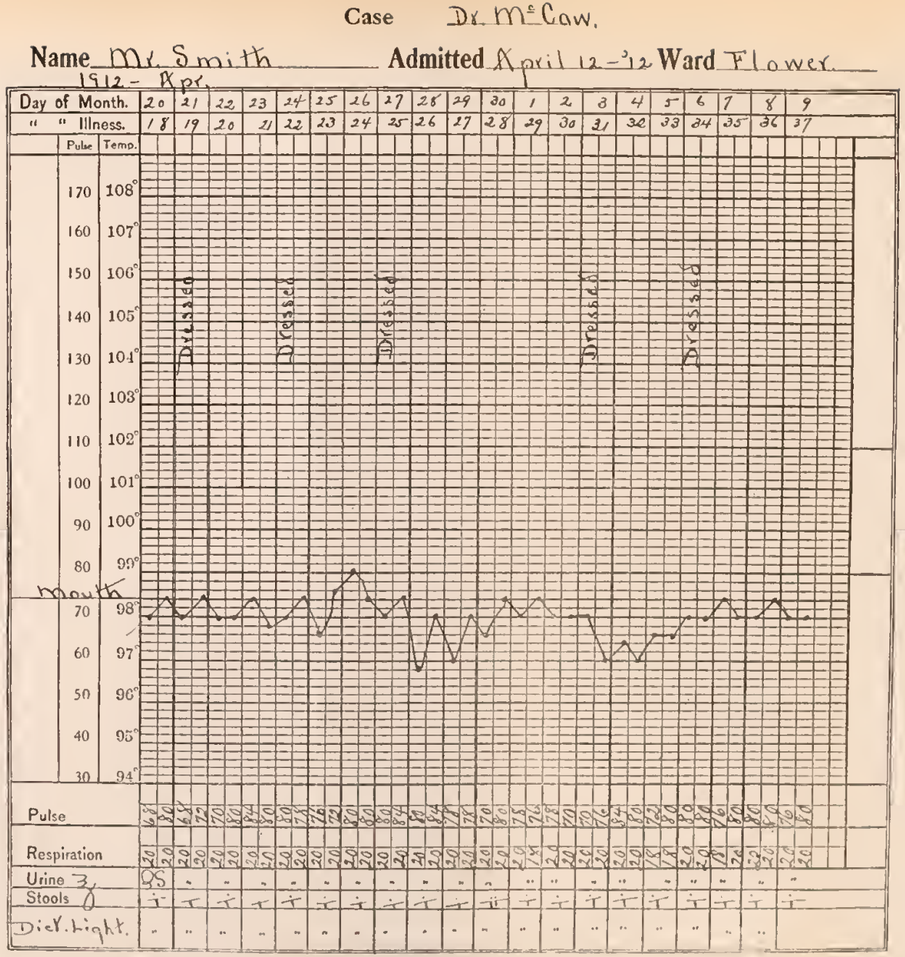
Patient, a woman 28 years old. *History*: Four years ago, deafness, tinnitus and full feeling in ears. Since this time deafness has fluctuated, with a total decrease in hearing. Several attacks of anemia and menstrual irregularities. *Examination* showed naso pharynx normal; eustachian tube normal; slight enlargement of one tonsil; nose negative; drum membranes normal; blush on the promontories; skin of forehead slightly thickened; hearing for watch, left ear, 2/60; right ear, 4/60; high limit of tone perception, as ascertained by Edlemann-Gaulton whistle, left ear, 2.7°; right ear, 1.5°; low limit of tone perception, left ear, 40 double vibrations, right ear, 32 double vibrations. The tuning fork was heard longer by bone conduction than by air conduction. *Treatment* consisted of internal administration of thyroid and ovarian substance. Vibratory massage was given around the ear and on the anterior triangles of neck. Nasopharyngeal applications of peroxide of hydrogen and saturated solution of feric alum. Mandell's solution was applied to tonsils. These applications were repeated once a week for six months. *Present condition:* subjective discomfort no longer noted in the ears. Menstruation is now normal. Inspection showed nose and pharynx negative; drum membranes normal; promontories pale. Hearing for the left ear, watch, 2/60; right ear, watch, 10/60; high limit of tone perception, by Edlemann-Gaulton whistle, left ear, 2.05°, right ear, 0.83°. Low limit of tone perception, left ear, 38 double vibrations, right ear, 28 double vibrations. Tuning fork in medium line by bone conduction referred to the left ear. A tuning fork of 96 double vibrations was heard in the left ear 49 seconds by air conduction and 64 seconds by bone conduction; in the right ear, this fork was heard 47 seconds by air conduction and 52 seconds by bone conduction.

OTITIC EXTRA DURAL ABSCESS AND SINUS THROMBOSIS. REPORT OF A CASE WITH REMARKS.

By JAMES F. McCAW, M. D., Watertown, N. Y.

The case which forms the subject of this report was that of a male, twenty-nine years of age, referred to me on March 16th, 1912. The following history was elicited: About one week before coming under observation he contracted a head cold, which was followed in three days by severe earache in the righ ear. This has continued ever since with increasing severity, so that he was unable to sleep for several nights. There has been no discharge and no swelling about the ear, but severe pain in and around it. Upon examination the canal was clear, the membrana tympani deeply congested with necrosis of the superficial epithelium. There was marked bulging of the membrana flaccida and Schrapnell's membrane; hearing was nil for the watch; no apparent swelling over the mastoid, but slight tenderness to deep pressure over almost the entire area; no point of special tenderness could be determined at this time. Physical condition excellent. The patient being from out of the city, was sent to the hospital, and a free myringotomy done. This gave him immediate relief from his suffering, and a profuse purulent discharge appeared, with the disappearance of all mastoid tenderness. The discharge gradually diminished and in ten days was very scant. The condition of the middle ear showed proportionate improvement, so the patient was allowed to return home with instructions for the care of the ear and to report in one week. Upon examination one week later, there was a profuse thick purulent discharge from the opening in the tympanic membrane; sagging of Schrapnell's membrane and the posterior superior canal wall; some tenderness over the entire mastoid area, but now there was extreme point tenderness over the region of exit of the emissary vein; temperature 99; pulse


Case Dr. McCaw.
Name Mr. Smith Admitted April 12-'12 Ward Flower
1912 April
Day of Month.
" " Illness.
Pulse
Temp.
Admitted 4 A.M.
To Op. Room 1 P.M.
Dressed
Dressed
Dressed
Dressed
Dressed
Mouth
Rectal
Pulse
Respiration
Urine
Stools
Diet-Light.
S.S.
Light


Case Dr. McCaw.
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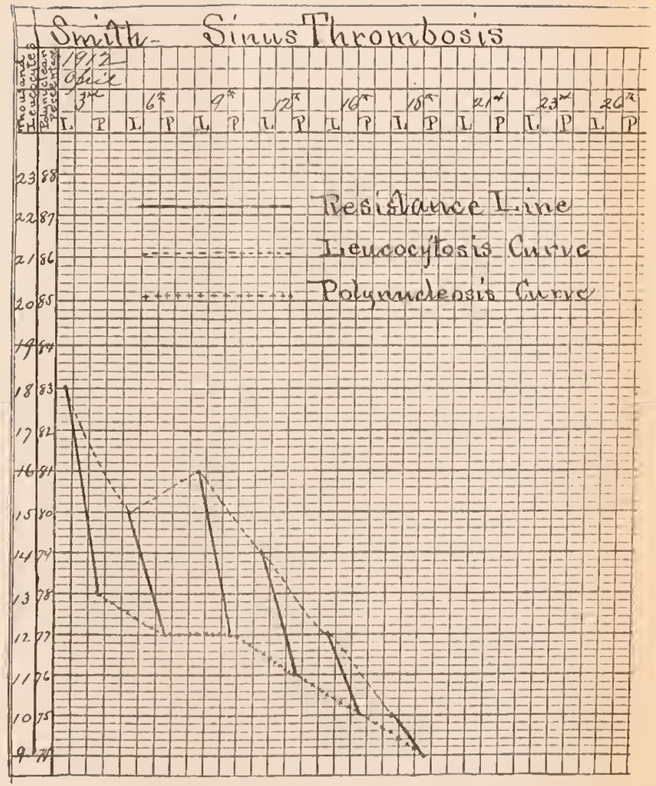
1912 - Apr.
Day of Month.
" " Illness.
Pulse
Temp.
Dressed
Dressed
Dressed
Dressed
Dressed
Mouth
Pulse
Respiration
Urine
Stools
Diet. Light.


Smith - Sinus Thrombosis
1912
April
Thousand Leucocytes
Polynuclear Percentage
Resistance Line
Leucocytosis Curve
Polynucleosis Curve

86; said he felt perfectly well except for the ear discharge. In view of the clinical manifestations, I insisted upon his going to the hospital for a mastoid operation. This was done the next morning, and on uncovering the mastoid, pus was not encountered until reaching the deep cells, where it was found the infection was expending its force. The bony wall overlying the Sigmoid portion of the lateral sinus was necrotic throughout its entire length. This was cut away, and just above the knee of the Sigmoid, an epidural abscess was encountered and about a half dram of pus evacuated; no granulations were present on the vessel wall. The abscess had exerted so much pressure on the sinus wall, that a clearly defined pit was left after evacuation. The sinus below this point was round, firm and resistant, without pulsation. At this stage of the operation, in view of my unsuspected findings, it was a question in my mind just what course to pursue. As this patient had given no clinical symptoms of Thrombosis of the Lateral Sinus, I determined to clean the area as thoroughly as possible, pack it as usual and watch developments. This was done. Examination of the pus showed a Streptococcus infection. A differential blood count was made every two or three days for nearly three weeks and I herewith present a chart of the findings, together with the temperature chart. This patient went on to a complete and uninterrupted recovery. The only unusual thing noticed in the healing process was, that the sinus wall, at its upper part, did not cover over until the other part of the wound was well filled with granulations.

There are several points of interest to me in this case. The first to impress itself: The impossibility of making a diagnosis of sinus thrombosis, before opening the mastoid, as there was absolutely no clinical evidence of sinus involvement, and not until the sigmoid groove was reached and the wall found exposed from necrosis of its bony covering, was it even suspected; although the findings showed a very extensive infective process around the sinus, with the accumulation of a fair sized epidural abscess which had made sufficient pressure on the sinus wall to leave a pit in it, even after

evacuation there were no clinical symptoms of absorption. This to the writer seems rather unusual. Again, was this an infective or non-infective thrombus? This question I am at a loss to answer. Whiting, in his most excellent monograph on sinus thrombosis, in speaking of the first stage says: "In this stage recovery is still possible, although improbable, *without operation upon the sinus*, the infective process occasionally resulting in a constructive inflammation, terminating in cicatricial obliteration of the sinus lumen, a conclusion fervently to be desired, but very seldom realized. The anticipation of such a favorable outcome is only to be entertained under most exceptional circumstances, namely; when the virulence of the infection is so far attenuated as to have nearly expended its energies during the invasion of the vessel walls and in the production of the resulting clot, and when the *residual activity* of the pathogenic organisms present in the thrombus is speedily destroyed by the germicidal action of the phagocytes and lencocytes. Under *no circumstances can such thrombi be regarded as non-infective;* that the extension to the sinus of an infective inflammation and the introduction into it of infective germs, should produce a non-infective clot, would be an anomalous state of affairs. That, however, the infection may be sufficiently attenuated, after producing the thrombus, to fail of causing disintegration of the same, in view of the action of the phagocytes and lencocytes, can be readily comprehended." On the other hand, Phillips, in his recent work on Diseases of the Ear, Nose and Throat, takes the opposite position and says, "In the absence of the classical symptoms of infection or thrombosis of the sinus, prior to operation, even though a perisinus abscess is discovered, it is inadvisable to explore it either by incision or by puncture, unless its walls are necrotic or gangrenous. Even if the surgeon is convinced that a clot is present, if no symptoms of infective thrombosis have appeared, it is inadvisable to interfere surgically with the sinus. The author is *firmly convinced that non-infective thrombi may develop in the lumen of a venous sinus,* which eventually become organized

into connective tissue. To operate upon cases of this type and thereby brave the danger of infecting the sterile thrombus is a questionable procedure."

The writer is not prepared to take issue with either of these authors as to the possibility of a non-infective thrombus in the presence of an infective area about it, but *we do think* that the patient's interest is subserved by not attacking the sinus surgically where there is an absence of clinical symptoms pointing to septic absorption; there can certainly be no harm in waiting until such evidence is present. Under close observation this can be detected at its inception, and the patient's welfare will be only slightly, if at all, jeopardized, with the possibility of an uninterrupted convalescence without further surgical interference. Frequent blood counts will render valuable assistance in not only showing the amount of absorption taking place, but is also a valuable aid in indicating the resistance of the individual. I am now having such examinations made in most of my cases, and am coming to place more and more reliance upon the findings. The outcome of my case at least warrants the procedure adopted.

Discussion.

Dr. McCaw, in closing the discussion, said he merely wished to emphasize the possible presence of non-effective clots, and to discuss the best way of dealing with them. The result in the case reported had certainly been most satisfactory.

ANTISEPSIS RATHER THAN ASEPSIS IN THE TREATMENT OF OTITIS MEDIA PURULENTA.

By SAMUEL THEOBALD, M. D., Baltimore, Md.

It has long been a matter of astonishment to me that in the minds of otologists of experience there should be any doubt as to whether antiseptic agents are or are not of value in the treatment of acute or chronic otitis media purulenta. In my own mind, certainly, clinical observation has left no room for such doubt. My conviction as to their efficacy is so clear, indeed, that in employing boiled water or a sterile wick to control a suppurating ear, I should consider myself only less censurable than in treating a gonorrheal conjunctivitis in similar fashion. When, therefore, I read in a very recently published treatise, devoted in great part to diseases of the ear, that in the treatment of otitis media purulenta the quantity of the fluid used, to insure a thorough washing away of all the pus, is more important than its chemical nature, and when I hear similar views advanced by otologists, as they often are, in formal and informal discussions, I wonder—well, I wonder if those who express such views have really made any comparative clinical observations of their own, or whether they have simply accepted someone else's, supposedly authoritative, dictum, or have merely been swayed by the fashion of the day.

Twenty-four years ago, when it was the custom to treat cases of acute as well as chronic suppurative otitis media by the insufflation, and even the packing of powders—boracic acid especially—into the external auditory canal, I read a paper before this Society[1] in which I advocated, as a safer and equally effective procedure, especially in acute purulent inflammation of the middle-ear, the employment of boracic acid in saturated solution, and in support of my contention reported a number of cases treated successfully in this way. Needless to say, the

1—Trans. Am. Otological Society, 1888.

discussion which the paper elicited was characterized by a considerable measure of scepticism.

Four years later, I presented to the Society another paper upon "The value of weak solutions of bichloride of mercury in the treatment of otitis media suppurativa",[2] and reported several cases in which, after boracic acid in solution had proved ineffectual, a bichloride solution, 1 to 8000, had promptly controlled the discharge, and brought about a closure of the perforation in the tympanic membrane.

In the years which have elapsed since this paper was written I have come to place chief reliance upon the mercuric solution, though cases have been met with occasionally in which the boracic acid solution has proved more efficacious, and it is upon the abundant experience of these twenty years that my faith in the value of antiseptics in the treatment of otitis media purulenta is based.

As to the method of employment, I may say that the boracic acid has been used in saturated solution, the bichloride, at the outset of the treatment, in the strength of 1 to 8000, though, not infrequently, this has been increased to 1 to 4000. If the discharge is copious, the ear is thoroughly but gently syringed, or douched, with whichever solution is employed, and, even at the beginning of the treatment, it has seldom been found necessary to do this oftener than two or three times daily.

When the discharge is scant, the ear is cleansed with the cotton mop; if the acute stage of the attack has subsided sufficiently to make it permissible, the discharge in the tympanic cavity is driven out by the Valsalva method, or by the Politzer bag; the ear is again wiped out, and a sufficient quantity of the warmed solution is poured into the ear to nearly fill the canal Before this is done the patient is placed in such a position, with the head inclined, that the ten minutes, at least, during which the solution should remain in the ear shall not prove too irksome. The solution is then allowed to run out, the ear dried, not very thoroughly, and the orifice of the meatus closed with a soft plug of absorbent cotton. How frequently this

2—Trans. Am. Otological Society, 1892.

application is made depends upon the effect produced, but it is never oftener than once in twenty-four hours.

I may add that I have felt no temptation to use the gauze wick that is now so much in vogue. If the discharge from the ear is profuse, the wick must unquestionably obstruct the outflow, unless it is changed more frequently than is possible except in hospital practice, with the services of a skilled assistant at command, while, if it is slight, it is not apparent how the wick can be of appreciable advantage. At all events, when efficient antiseptics are used the need for it is not evident.

When, formerly, I employed boracic acid more often than I do now, I found, as the case progressed towards recovery, that its too frequent use was detrimental, since, by keeping the parts too dry and the tympanic membrane too anæmic, the healing of the perforation in the drum-head was retarded or completely arrested; but this does not seem to happen when the bichloride solution is used.

There are few cases, even of chronic otorrhœa, that seem to me to call for other treatment—apart, of course, from operative procedures—than the employment of the bichloride solution in one of the ways indicated; but, exceptionally, especially when there is extensive destruction of the drum-head, the insufflation of boracic acid or of boracic acid and aristol or oxide of zinc, equal parts, has been found efficacious when the mercuric solution has failed.

I could exhaust your patience by citing many cases from my clinical records in proof of the efficacy of the treatment I have outlined; but I shall content myself—and am sure of your approval in doing so—with a brief account of a single typical and very apposite case which recently came under my care.

A Baltimore lad, at school in New England, had an acute otitis media, for which he was sent to a specialist in Boston. The membrane was incised, and directions were given that the ear should be syringed with "water," (doubtless boiled water) only. After eight days of this treatment, as the discharge continued, he was brought home, and came at once to see me.

The canal was found to contain a considerable quantity of pus, and the drum-head was so opague and sodden as scarcely to be distinguishable. The pus was wiped out with a cotton mop, and bichloride solution (1 to 8000) instilled, and allowed to remain in the ear for ten or twelve minutes. The ear was then dried, and closed with a cotton plug, as has already been described. Syrup of the phosphates of iron, quinine and strychnine, in drachm doses, was also prescribed. The next day the ear was clean and dry, and already the appearance of the membrane had changed greatly for the better. The bichloride instillation was repeated, and the patient was directed to resume the use of a nasal spray—bichloride of mercury in normal salt solution, 1 to 5000—which I had prescribed for him several months previously. On the following day the drum-head was beginning to present a fairly normal appearance, though still hyperaemic, and Valsalva inflation gave a distinct, dry, non-perforation sound, after which hearing for whispered words was found to be practically normal. As a matter of precaution a final instillation of the mercuric solution was made.

Two days after this, though the membrane was less hyperaemic and hearing for the voice normal, he reported having had some twinges of pain in the ear since his previous visit. For this, the solution of the alkaloids of atropia and cocaine in oil of sweet almonds,[1] which I suggested many years since as a useful remedy in the early stages of otitis media, was prescribed, eight drops in the ear three times a day.

Two days later, there having been no recurrence of pain, the ear was in such "good shape" that the patient was given permission to return to school.

Could there be a more striking refutation than this case affords of the teaching, so prevalent at the present day, that it is the quantity of the fluid that is thrown into the ear, not its chemical composition, that is of importance?

As a final observation, I may add that since the great majority of cases of acute otitis media, if not purulent from the outset, becomes so, in spite of aseptic precautions, after the

1—The formula is: Atropiæ (alk.) gr. j; Cocaine (alk.) gr. ij; ol. amyg. dulc. dr. ij.

membrane is incised or a spontaneous rupture has occurred, it is, in my opinion, wise, in all cases, to begin the antiseptic treatment without loss of time or waiting for further developments.

Discussion.

Dr. John F. Barnhill, of Indianapolis, Ind., said he had been taught that very little antisepsis could be obtained in the middle ear through an ordinary perforation or paracentesis incision. He had tried in all sorts of ways to get fluid into the middle ear cavtivity through such openings in the drumhead. but he had been unable to do so. He had been in the habit of teaching his students that not a drop of fluid gets into the middle ear when it is merely dropped into the outer canal. With air-pressure or some other method fluid could be forced in under ten or fifteen pounds pressure. Its entrance into the middle ear cavity under such circumstances was proved by the fact that some of the fluid passes into the Eustachian tube, and the patient complains of it. The improvement in the case reported by Dr. Theobald was remarkable. In most cases of middle ear when it is merely dropped into the outer canal. there was extension to the antrum and mastoid attic. According to Politzer pus had been found in the antrum in nearly every post mortem examination of patients dying while having middle ear suppuration. Unless some force was used the speaker doubted if fluid ever reached the mastoid antrum and cells. It seemed to him quite as wise for the urologist to attempt to cure abscess of the kidney by dropping fluid into the uretar as it would be for the otologist to try to cure disease in the mastoid antrum by dropping fluid into the middle ear. He preferred the wick treatment to simple instillation.

Dr. E. Terry Smith, of Hartford, Conn., has used for an anesthetic in paracentesis for otitis media a mixture composed of the crystals of menthol, cocaine and carbolic acid. These are rubbed together and they make an amber colored fluid which when applied to the drum produces a complete anesthesia and consequently renders the operation painless.

After using this it has been noticed that the number of cases that go on to recovery with simply a serous discharge, have almost doubled.

Dr. Welty, of San Francisco, Cal., had used the treatment suggested in the treatment of acute otitis media not dependent upon infectious diseases, but he had not the good results to which the speaker referred. Many of his cases had gone on from four to six weeks before recovery. He had adopted the plan of using the intratympanic canula when it could be employed. Medicines put into the canal were of little use, only those put into the tympanic cavity being of avail. If adenoids and tonsil were removed before the case had lasted three days, in the majority of cases the ear would be well before the throat had healed. He had yet to see anything serious result from such procedure. Furthermore, I believe that an ear that discharges for more than six or eight weeks should be subjected to the acute mastoid operation, because of the fact that the hearing will be impaired if the discharge is allowed to continue; while if they are operated before such impairment they will have a good ear. He advocated the treatment of chronic otitis media by intratympanic irrigation with various antiseptic solutions and drying covety.

Dr. William C. Braislin, of Brooklyn, N. Y., had never used bichlorid in the middle ear because it seemed to be irritating, but he had used it in furuncle. He employed it very freely in a strength of one to four or five thousand, warm, five drops every five minutes for an hour or two, two or three times a day. Very prompt relief of pain and rapid recovery follows its use.

Dr. B. Alexander Randall, of Philadelphia, said he had been taught by Risley in '80 that bichlorid was one of the most valuable agents, in ear or eye. Where irrigation does not carry the fluid into the tympanic cavity, it was simple enough to obtain penetration by pressure, that of the finger-tip being sufficient to carry the fluid not only into the tympanic cavity but into the pharynx, where he sometimes hesitated to use

bichlorid for fear too much might pass and cause intestinal trouble. Cases of the acute form cited required a certain length of time to bring them to a finish. They ran, as a rule, a fairly definite course which could not be shortened or aborted —not many of them could be checked in the first week. In serious cases this was possible. As to the matter of penetration with the intratympanic canula or with the mere forcing through of the fluid with peroxide he had had patients froth at the nose, which proved the unmistakable penetration, tho, he did not advocate this procedure. A greater success was assured if the fluid was used very hot, patients being able to bear a temperature of 115 to 116 degrees Fahrenheit. He wished to make a protest against the old-fashioned habit of speaking as though the antrum and attic are not parts of the tympanic cavity, emphasizing the point that the antrum is the posterior portion of the tympanic cavity.

Dr. James E. Logan of Kansas City, Mo., thought the question of treatment of Otitis Media would be very much simplified if Otologists would give more attention to the *cause* of the disease. It is well enough to use irrigation or other methods of cleansing the external ear; but it is also most important to relieve the cause of the trouble.

Acute inflammation in the vault of the pharynx extending up through the Eustachian canal is the cause of most of these middle ear conditions. Keep the external ear clean by irrigations of Boracic Acid solution, at the same time relieve the inflammation in the vault of the pharynx. In some cases the application of a 60% solution of Nitrate of Silver to the vault of the pharynx, together with the cleansing of the external canal is sufficient.

When Adenoids are present he often removes the Adenoid during the existence of the middle ear inflammation and often the ear condition recovers very rapidly.

Dr. S. MacCuen Smith, of Philadelphia, quoted a well-known pathologist as saying that bichlorid of mercury and nitrate of silver do nothing more than coagulate the albumin, underneath which various forms of germ life flourish. The

speaker advocated changing the treatment of suppurative otitis media, from the fact that one micro-organism would thrive in the presence of a certain drug which would destroy another. He had used tincture of iodin in various strengths, in connection with other drugs, with good results.

Dr. Hiram Woods of Baltimore, called attention to the well-known class of cases in which thorough cleansing of the ear, once or twice, is followed by recovery. It was not scientific to attribute such a result to boric acid or any other agent. mere removal of the detritus apparently being sufficient. Referring to the wick treatment mentioned by Dr. Barnhill, the speaker thought it would be interesting to know if the members of the Society were opposed to this method. He had found that, after incision of the drum membrane for acute otitis media, unless the discharge is very excessive, a small wick of gauze saves a good deal of syringing and danger of re-infection. Convalescence was sometimes shortened by this treatment.

Dr. Theobald, in closing the discussion, said it could be demonstrated, in the majority of instances, that fluid used as described does reach the middle ear cavity. When the swelling and inflammation become less marked, patients not infrequently, indeed, complain of the taste of the solution. Bichlorid in the strength employed is not irritating, as suggested by one of the speakers. One to eight thousand bichlorid in acute cases, and one to four thousand in sub-acute and chronic cases, causes no irritation whatever. Referring to the suggestion of using a stronger solution for the purpose of controlling furunculosis of the external auditory canal, he said ophthalmologists generally use yellow oxid of mercury for this purpose. Dr. Randall need not feel anxious about the toxic effect of bichlorid getting into the pharynx through the Eustachian tube. If half a dram of the 1 to 4000 solution should pass into the pharynx the patient would swallow only about 1-130 of a grain of bichlorid. The speaker had had no experience with the tincture of iodin in otology. All ophthal-

mologists are familiar with its value in the treatment of corneal ulcers. Simple cleanliness, as suggested by Dr. Woods, doubtless gives good results in some cases, but a larger percentage of cases recover promptly with antisepsis.

EXTERNAL RADICAL OPERATION FOR CHRONIC EMPYEMA OF THE AIR SINUSES BY THE ORBITAL ROUTE.

W. SOHIER BRYANT, A. M., M. D., New York, N. Y.

Chronic purulent pansinusitis of the accessory sinuses of the nose is generally admitted to be an affection very resistant to treatment of any kind and practically incurable by any other than surgical means. Because of peculiar development or because of the extent of the involvement, the surgical access to these diseased cavities lying below the base of the brain at times requires an opening through the upper face which sometimes results in great disfigurement.

Etiology: Chronic purulent pansinusitis is secondary to neglected acute purulent infections of the nose.

Diagnosis: The diagnosis should be established by the help of a skiagram. There should be an intranasal inspection which, in this condition, will show pus coming from the orifices of the sinuses and a granular hypotrophic condition of mucous membrane. Transillumination discloses more or less marked opacities in the special areas of the sinuses in proportion to the amount of pus present; and internal and external palpation reveal an oedematous condition and hypersensitive areas.

The diagnosis of chronic purulent pansinusitis is easy in cases where the process is advanced enough to be associated with intense continuous or intermittent headache and abundant purulent nasal discharge. In cases where pain and discharge do not drive the patient to seek relief, the condition may continue unsuspected for years, in spite of grave constitutional symptoms, which indicate toxic conditions with functional nerve disturbances, and cranial, heart and lung difficulties. The advance of the process is indicated by severe pain or grave intracranial symptoms.

Surgical Treatment: Our effort in the operation for pansinusitis is to use the technical procedures which give the best results pathologically, functionally and cosmetically. Pathologically, all diseased surfaces must be changed into healthy ones. Pathological tissues such as pyogenic membranes and granulation tissue obstructing the drainage of the sinuses, are to be removed. Functionally, the eye must be restored to its normal position and function, and the nose must again become a moist passage with olfactory sensibility and must contain erectile turbinal tissue. Cosmeti-

Fig. 1. Skull marked to show line of incision. X—Skin incision. XX—Periosteal incision. O—Bone incision.

cally, there must be no external scar and the lines of the face must not be altered by frontal unevenness and depression.

Surgical treatment is directed to drainage of the sinus affected and removal of pathological tissue. The external route is chosen in preference to the intranasal route in the cases where the bone conformation does not favor intranasal drainage, in cases where the disease is of very long standing and is far advanced, and especially in cases with dangerous intracranial complications.

These ends are well accomplished by the following technic. General anaesthesia is administered by insufflated vapor through a mouth tube.

Anatomical Outline.

The usual curved line incision is made through the skin in the center of the eyebrow and down the side of the nose. The cut is carried outward as far as the outer lateral wall of the frontal sinus. Nearly parallel to this cut but lower down, the periosteum is incised on a curved line which passes along the upper orbital surface and goes farther back, as it passes down the side of the nose. The periosteum is carefully loosened; and lifted with it, is the pulley attachment of the superior oblique muscle, and the free part of the lachrymal sac. The eye is retracted by gently drawing the angle of the skin flap downward and outward with a vulsellum forceps.

The bone is opened through the frontal bone at the inner edge of the orbit. The line of bone incision should, if possible, be prevented from crossing the line of the periosteal incision. The object of making these skin, periosteum and bone incisions on separate levels, is to prevent the stretching of the scar. This technic assures no scar if the sutures hold. Enough of the orbital plate of the frontal bone should be removed so that the frontal sinus may be opened to allow thorough curettage.

We should also remove a part of the nasal process of the superior maxilla, a part of the adjacent nasal bone, as much of the ethmoidal orbital wall as seems necessary to remove all diseased tissues, and the upper part of the plate of the lachrymal bone, without injuring the lachrymal sac. The infundibulum should be enlarged as wide as the conformation of the parts will allow and the middle turbinate and ethmoidal cells should be removed by the aid of clear illumination. Before attacking the ethmoid directly, the procedures are directed to a completion of the frontal sinus work in order to minimize the possibility of post operative meningitis through injury of the inner table. This is done to prevent infectious material from being car-

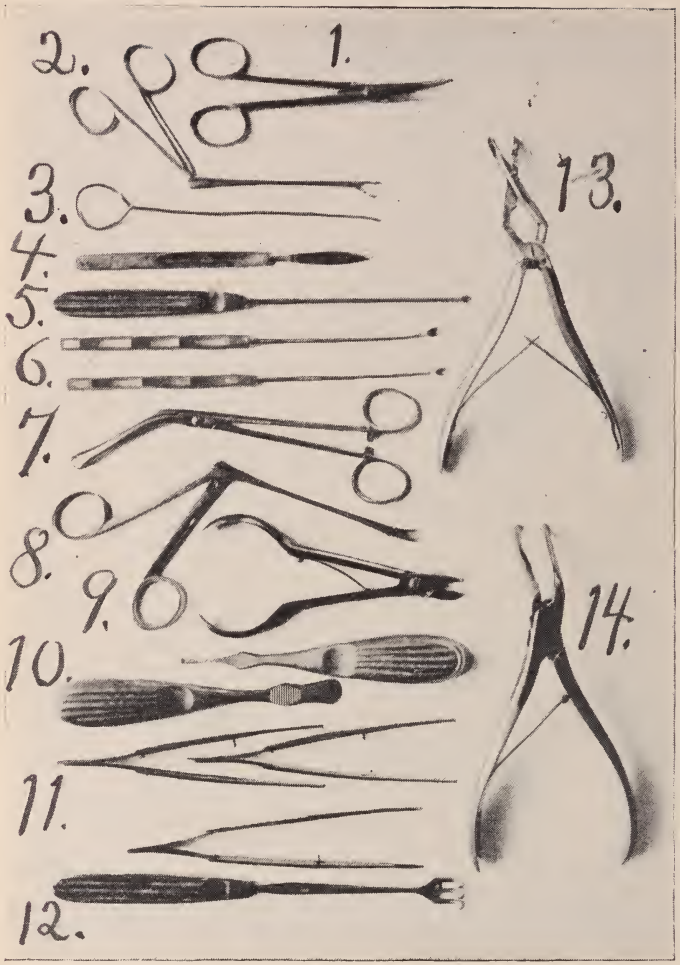


Fig. 2. Instruments used in the operation beside needle and needle holder. (1) Scissors. (2) Alligator forceps. (3) Probe. (4) Scalpel. (5) Forward cutting curettes. (6) Sidewise cutting curettes with malleable handle, different sizes. (7) Vulsellum forceps, to retract lower flap. (8) Alligator cutting forceps. (9) Short rongeur. (10) Periosteum elevators, different sizes. (11) Forceps, dressing and mouth-toothed. (12) Retractor for upper flap. (13) Punch forceps. (14) Long-beaked rongeur.

ried from a dirty frontal. The excavation proceeds till all cells have been opened and trabeculi removed. To insure rapid convalescence and to avoid recurrences, this complete removal of cells is as necessary in sinus work as in work on the mastoid cells.

The sphenoid sinus is next opened and last the maxillary antrum is widely opened above the lower turbinate. These openings are thoroughly curetted and the edges trimmed with alligator punch forceps.

The special instruments used are a medium-sized curette with a long malleable shaft for making the initial opening in the orbital plate of the frontal, and a small rongeur to enlarge this opening; the same curette and rongeur are used for cleaning out the frontal sinus; alligator punch forceps are used for removing middle turbinate. The trabeculi of the ethmoid are broken down by the curette and trimmed by the alligator forceps. All ethmoidal cells must be broken down in order to assure intranasal convalescence. The curette enters and breaks the wall of the sphenoidal sinus and the alligator forceps trims them smooth. The maxillary sinus is opened with the same curette; this sinus is cleaned out and the edges are trimmed with alligator punch forceps.

During the operation, bleeding is controlled with gauze packing and adrenalin, applied for a few moments as needed. When it is impossible to remove all diseased tissue, a drain is left in at the inner orbital angle. The wound is sutured by interrupted No. 00, ten-day chronicled catgut for the periosteum and No. 00 black silk for the skin; a bandage is put across the forehead and a compress over the eye. No dressing is placed in the nose or sinuses or on the external wound, which is bloodless.

Post operative bleeding is rarely of any moment. If, however, bleeding necessitates post-operative packing, this packing is done with gauze strips placed against the bleeding surfaces through the nose. There should never be indiscriminate packing of the whole cavity.

The advantage of this technic is that no scar remains. There is no possibility of deformed outlines, because of the

primary union of the skin wound and periosteum and because of the preservation of the vertical plate of the frontal bone. Since the operation obliterates all the ethmoidal cells and drains the major sinuses, the convalescence is rapid and there are no recurrences.

Post operative treatment is directed to keeping up drainage by preventing the occlusion of the orifices of the sinuses which are necessarily enlarged to their maximum by the operation. Crusts should be softened with hydrogen peroxide, and if granulation tissue is troublesome, it should be removed with forceps. Irritation of the conjunctival sac should be controlled by the use of appropriate colleria. Occasionally a mild boric collerium or argyrol solution is indicated for use in the eye for a few days. A hydrogen peroxide or oil spray is often needed in the nose to remove exudative accumulations until the mucous membrane has completely covered the denuded surfaces.

Success in this operation depends in large part upon the eradication of all ethmoidal cells, upon obtaining sufficiently large infundibular passage, upon the maintenance of free communication and drainage from all the sinuses, and upon primary union of the external wound.

The surgical technic prescribed for operation on all the sinuses is readily modified to apply to the following groups of sinuses:

(1) Frontal, ethmoid and antrum.
(2) Ethmoid, sphenoid and antrum.
(3) Ethmoid and antrum.

The following example illustrates the indications, procedures and results of the operation:

Patient: Woman aged 40 with chronic purulent pansinusitis. *History*: At six years of age patient had scarlet fever and the trouble in the head began then. Since then pus had run from left side of nose. When changing the position of the head, this discharge was at times extremely abundant. Had pain in the left side of nose, over left eye, sometimes in vertex and sometimes in back of head. Intense

pain in the head began when the patient had diphtheria at 16 years of age.

There is 24 years of definite history of frontal sinus disease following the attack of diphtheria. Fourteen years ago, at 26 years of age, the patient had grippe and intense pain in left eye. At twenty-eight years of age, 12 years ago, she had a severe attack of erysipelas of head and shoulders. Temperature of 105° for nine days. She had phlegmona beginning on top of nose, preceding and after the attack of erysipelas. Has been treated in various cities, then and since. At about this time, 12 years ago, the pa-

Fig. 3. Line of incision. Fig. 4. Patient after operation.

tient was not expected to live, was in a comatose condition (probably meningitis), not typhoid. No definite diagnosis was made at the time. Patient states that this condition lasted some time.

At time of operation the patient had intense pain in left eye, and complained of loud pulsating tinnitus in left ear and of purulent dactryo-cystitis.

Examination-Inspection: Tip of nose phylnjectatic. Right side of nose negative. Left nasal fossa open, mucosa granular and bathed in pus, which came chiefly from the middle meatus. Much discharge down back of naso-pharynx. Pulse, respiration and temperature negative. *Palpation:* Marked tender-

ness about inner cantus of the left eye, on orbital plate of frontal bone and above middle meatus in the left nasal fossa. *Transillumination:* Right side negative. Left side: Eye region dark and antral region dark. Showed a large frontal sinus with blurred outline and no boundaries were visible. *Skiagram*: Left antrum dense, as if filled with pus. Left ethmoidal and sphenoidal sinuses showed slight increase of density. Left frontal sinus was more obscure than right. *Diagnosis*: The above findings indicated sinusitis of frontal, ethmoid, sphenoid and maxillary sinuses. *Operation*: Skin was sterilized with Harrington solution. Anaesthesia, gas-ether 1½ hours. Technique followed as described above. The mucosa of the frontal sinus was very thick; sinus empty; mucosa of antrum and ethmoids thick; posterior ethmoidal cells contained pus; antrum full of pus. The mucosa of the sphenoid thickened and a little pus was found. Periosteum was sutured with 10-day chromicized catgut No. 00. Interrupted skin suture No. 00 black silk for skin.

Convalescence: *Day following operation* there was some oozing from nose and down throat. Ciliary region of left eye echimotic. Relieved some headache by morphine, ¼ Gr., given hypodermically. The temperature reached 100 2/10° Fahrenheit, Rectal. It did not again rise above 99°. *Second day*, both eyes closed by oedema and echimoses about both eyes Boric acid collerium was used for left eye, which showed some conjunctival irritation and was slightly purulent. *Third day*, removed superficial suture about eye. Took dressing from left eye. Patient had sharp pain on using eye, and diplopia was marked. Much pain in attempting to open left eye. Some pain over top of head. Patient up and dressed. Left nares rather dirty and considerable swelling. Until now the discharge has been clean. Hydrogen peroxide with applicator and oil spray. *Fourth day*, all skin sutures removed. Hydrogen peroxide and oil spray in nose. *Fifth day*, patient feels very well. Oedema nearly gone on right side. *Seventh day*, some occipital pain, but no other pain. Patient can open left eye. Nose clear and pretty clean; hydrogen peroxide and oil spray. *Tenth day*, no pain, nose felt perfectly comfortable;

patient felt well. A little pus from anterior superior region, probably from antrum. Used 10% silver nitrate solution on cotton applicator about nose. *Twelfth day*, blowing nose hard had caused a little emphysema about the wound. *Fifteenth day*, nose crusty; wiped out. *Sixteenth day*, nose clean. *Thirty-eighth day*, left eye restored to alignment. *Second month*, patient florid, has gained weight. (Since operation has had at times various pains but apparently of no consequence). Hearing of left ear has improved very much. Still slight pulsating tinnitus. *Fourth month*, still a slight amount of purulent nose discharge. *Seventh month*, owing to the persistence of this slight purulent discharge from left antrum it was necessary to remove a little granulation tissue with alligator punch forceps, under cocaine anaesthesia. Temperature remained normal after this operation. Four days later, everything was in good condition in eyes and nose. No scar remains. The patient has no more tinnitus. A normal musoca seems to have been finally established in the nose. No syringing or washing was used at any time by the author, but powders, wiping or sprays were used. Patient has been under observation 1½ years and remains in good condition.

Summary: The important points of this operation are.

(1) The use of the over-lapping flaps.

(2) Complete removal of orbital plate of frontal bone and orbital plate of ethmoidal bone.

(3) Enlargement of the infundibulum as wide as the lachrymal duct will allow.

(4) Complete removal of all cells.

Discussion.

Dr. Thomas J. Gallaher, of Denver, Col., said that the operation which Dr. Bryant performed was the lower portion of the Killian operation. He complimented the author in his endeavor to save the anterior wall of the frontal sinus. The speaker had advocated the preservation of the anterior wall three years ago in a paper read before the Western Section of the American Laryngological, Rhino-

logical and Otological Society, at Colorado Springs, Colorado. He is of the opinion that while the anterior wall cannot be preserved in all cases, it certainly can be done much more frequently than is now attempted. The speaker employs the internal operation first, proceeding to the external operation when the first is not satisfactory, especially when there is continuous pain in the head with discharge from the frontal sinus. He warns against the possibility of fracturing the Os Planum and infecting the orbit when the burr or rasp is used. He cited a case in which he had accidentally fractured the Os Planum with the rasp. Orbital cellulitis ensued, necessitating the immediate performance of the external operation. The patient made an uneventful recovery. He uses no drain externally and immediately closes the wound with horse-hair sutures, which may remain in place for a week. At the end of the first twenty-four hours and on every day thereafter, as long as is necessary, he applies intranasally a solution of nitrate of silver, fifty grains to the ounce, to keep down the granulations. He instills a few drops of one per cent. solution of homatropin into the eye daily for five days. This can be more quickly neutralized than atropin by means of eserin in case of increased intra-ocular pressure.

Dr. Charles W. Richardson, of Washington, D. C., understood that the point to which Dr. Bryant called particular attention was the making of a different line of incision in the periosteum from that employed in the Killian operation. He emphasized the fact that the region involved in the operation under discussion is a difficult one in which to work, and one which requires due caution in the character of the technic employed. He wished to protest against the internal operation as employed by Dr. Gallaher. Fracture of the Os Planum was an accident which occurred more frequently than most otologists were willing to acknowledge, and was doubtless the cause of many cases of meningitis. He deprecated the making of a pocket-like wound in an unprotected surface as in the simultaneous performance of a sub-mucus

septal operation and an ethmoid or antral with its presence of bacterial laden pus and nasty bone disease.

Dr. B. Alexander Randall, of Philadelphia, called attention to a paper on this subject read before the American Medical Association, in 1893, by Knapp, who gave all of the details except this skin flap and a few other points. The matter was very well put forward by Killian later, but his work was peculiar mainly in removing the external table of the frontal bone and preserving the brow-ridge.

Dr. Bryant, in closing the discussion, said that the technic of the operation described in the paper was more like that described by Dr. Knapp than any other he had been able to find in the literature on the subject. The lower wall of the frontal sinus and most of the inner wall of the orbit were entirely removed. A cautious intranasal operation should be performed first, if possible, and if successful, nothing more is necessary. The intranasal operation on the ethmoid cells is a dangerous procedure, with a high mortality. Complete exenteration of the ethmoid is especially a dangerous procedure. The trochlear of the internal oblique is always detached; otherwise it is impossible to reach the field of operation. No permanent interference with the motion of the eye resulted from the operation.

HYSTERIA SIMULATING MASTOIDITIS WITH SUSPECTED BRAIN ABSCESS FORMATION.

By S. MacCUEN SMITH, M. D., Philadelphia, Pa.

Patient, H. T., colored, aged 26 years. Occupation, porter. Seven months prior to his appearance at the Jefferson Hospital Clinic, the patient was hit by a baseball over the mastoid process on the right side, the blow being followed by bleeding from the right ear and unconsciousness for twenty-four hours, after which he was totally deaf in the right ear, and so deaf in the left that one had to speak in a loud tone, close to the ear, to make him hear conversation. The hearing in the left ear, however, was restored within a short time.

There was excruciating pain over the right mastoid process, extending from the occipital to the parietal region, and continuing more or less severe during the entire seven months. After the accident the patient suffered from constant buzzing tinnitus aurim and throbing pain in the right ear. He also had periodic epistaxis, increased when lying down, and frequent attacks of vertigo, so severe that to prevent falling he was obliged to assume the recumbent position. He never had any attacks of unconsciousness after the one immediately following the accident. The patient also suffered from complete left hemianesthesia, in proof of which a pin could be run into his flesh at any point on that side without apparently causing pain.

Examination showed the ear to be practically normal in all respects, save the apparent severe pain over the mastoid process and temporal region, both of which were markedly increased by even slight pressure. The external auditory canal, membrana tympani and ossicles, in so far as could be determined by a physical examination, were normal. In the right ear there was complete absence of both aerial and bone conduction, and we were unable, during frequent and varied tests, to change this conclusion.

Blood examination: This report shows a moderate leukocytosis (14,400), with an increase in polymorphonuclears (84%).

Ophthalmological examination: The report shows the vision in the right eye 20/50 plus, the right nerve head markedly hyperemic, and the physiological cup filled in. The rest of the fundus is normal. There is a simple hyperopic astigmatism of 1.00 D, which no doubt accounts for his poor vision. The vision in the left eye is also 20/50, the nerve head is similarly hyperemic and the cup filled in. There is no gross lesion. Simple hyperopic astigmatism, 1.00 D. The hyperemic nerve head stands out in marked contrast to the surrounding fundus, which is typically negroid.

X-Ray report: Mastoid cells are of small area, but the individual cells are large and there is some bone defect in the upper portion. The process is in no part sclerotic. The lateral sinus is somewhat superficial. There is no positive indication of an intra-cellular exudate, but the shadow is suggestive of this condition.

Neurological examination, by Dr. George E. Price, shows gait normal, with eyes open, slight ataxia with eyes closed. Station, somewhat plus sway. Ataxia of upper extremities questionable. Pupils equal and normal in reaction; no nystagmus nor external ophthalmoplegia. When first seen, the musculature of the face was apparently normal; when examined the following day, there was slight drooping of the mouth on the left side; muscular movement normal, however. Movements of upper extremities normal. Abdominal and cremasteric reflexes present. Left leg weak as compared with the right leg. Knee jerks plus on both sides. No ankle colonus on either side, although a pseudo-colonus was at times obtained. Babinski's sign absent. There is a diminution of sensation over the entire left half of the body.

While hysteria was discussed as a probability, the fact that the ear symptoms were in one side and the sensory and motor symptoms on the opposite side, together with the blood findings, led to a diagnosis of abscess of the right temporal lobe.

The temperature and pulse, both pre-operative and post-operative, were practically normal. This, of course, would

not bear out our suspicion of brain abscess formation, and yet, when we consider how frequently the classical pressure symptoms, as indicated by lowered pulse, respiration and temperature, fail in the presence of brain abscess, we were probably justified in suspecting that condition, after a careful consideration of the history of trauma, the presence of hemianesthesia, the eye report, the nervous phenomena present, and the blood picture.

Five days after the patient was admitted to the hospital, a simple mastoid operation on the right ear was done for exploratory purposes. After the removal of a very hard cortex no actual disease was discovered in any part of the cavity, though the tip of the mastoid bled more than normal. One day after the operation, the hemianesthesia cleared up, the vertigo disappeared and the hearing became nearly normal, in the right ear, having been previously regained in the left, as above stated.

This patient was seen quite recently, or about four months after the operation, and the improvement incident thereto is still maintained, which clearly shows that we were wholly deceived by a typical neurasthenic, and although the patient might have recovered under non-surgical psycho-therapeutical treatment, as was probably indicated by the preponderance of subjective over objective symptoms, yet we felt justified in performing an exploratory operation for the reasons above stated, and ordinarily, if the patient had suffered from an otitis media, these symptoms taken together would offer a complete picture for operative intervention for mastoid disease, and the findings at the time of operation would have enabled us better to determine the presence of an intracranial lesion.

PHYSICAL ECONOMICS—WITH REFERENCE TO THE ECONOMIC VALUE OF MAN AND THE MEASUREMENT OF DAMAGES TO ANY PART OR PARTS OF HIS BODY, BY STANDARDS OF MEASUREMENT AND MATHEMATICAL FORMULAS, IN A MANNER EQUITABLE TO ALL CONCERNED.

By ERASTUS EUGENE HOLT, A. M., M. D., LL.D., Portland, Me.

Any physician who devotes more or less of his time to the treatment of diseases of the ear may be called upon to give expert testimony as to the damages to the ears, from injury or disease, either alone or in connection with damages to other parts of the body. For this reason there should be some standard method by which all those who are called upon to render this service may arrive at definite conclusions which will agree with all the existing conditions of the person who has suffered such damages.

The aim of this paper is to present a standard method of procedure which may be employed by any physician who may be called upon to render such a service. By such a method definite conclusions may be reached, and although the conclusion reached by one physician may differ from that of another in a given case, nevertheless these conclusions will have been reached by the same method, and this difference may be readily accounted for by examining the several factors employed and noting the different values given to these factors. Thus every step of the process of each independent observer may be taken up separately, compared with one another, and discussed. Each may state his reasons for determining the value given to each factor in the problem. If an agreement cannot be arrived at in regard to the value given to each factor, a note may be made of this disagreement and this difference in the values of the factors, will indicate precisely why the results differ from one another. Thus all physicians who are called

upon to determine damages to any part, or parts, of the body, from injury or disease, will be employing one uniform method corresponding precisely to that by which the value of any physical force is obtained. This method is known as the natural science method. This method was used by the scientists when they devised a way to measure electricity and defined the unit of electro-motive force to be a volt, named in honor of Volta, an Italian physicist, which consists of one ampere, named in honor of Ampere, a French psysicist, with a resistance of one ohm, named in honor of Ohm, a German physicist. These three elements when used as factors and multiplied together are equal to a watt, named in honor of Watt, the great English inventor. America, the home of some of the greatest inventors is not represented. If we use the first letter of each of these words as a symbol to represent them as factors in a formula, we have as follows: V x A x O=W, 746 of which watts equal a horse power. A horse power was designated by Watt, when he invented the steam engine, to be equal to a force which will lift 33,000 pounds one foot in a minute.

With reference to the economic value of man, we find that in the seventeenth century Sir William Petty was the first to attempt to determine the economic value of man from his earning ability. In the latter half of the same century, Adam Smith of Scotland, in that classical work entitled "Nature and Causes of the Wealth of Nations," compasses the whole subject in four sentences, as follows:

"When any expensive machine is erected, the extraordinary work to be performed by it before it is worn out, it must be expected, will replace the capital laid out upon it, with at least the ordinary profits. A man educated at the expense of much labor and time to any of those employments which require extraordinary dexterity and skill may be compared to one of those machines. The work which he learns to perform, it must be expected, over and above the usual wages of common labor, will replace to him the whole expense of his education, with at least the ordinary profits of an equally valuable capital. It must do this too, in a reasonable time, regard being had to the very uncertain duration of human life, in the same manner as to the more certain duration of the machine."

Since that time scores of writers have devoted more or less time to the subject. It remained, however, for William Farr, in the nineteenth century, who spent his life in the Registrar-General's office of England, writing on vital statistics and allied subjects, to become an authority on these subjects throughout the whole civilized world. Dr. Farr comprehends this subject in one sentence as follows:

"The value of any class of lives is determined by valuing first, at birth, or any age, the cost of future maintenance and the value of future earnings."

Ernst Engel of Germany in a work published in Berlin in 1883 upon this subject, for the extracts from which I am indebted to Professor Wilcox of Cornell University, computed the cost of the production of human beings from household account books. He was not inclined to accept Farr's method, not however giving any reason therefor. Engel, after an elaborate consideration of the subject, developed tables of the value of human beings from the point of view of the cost of production. He, however, omitted to reckon one of the chief factors in the production of a human being, namely, the value of the services of the mother. When these services of the mother are added to the cost of production which he obtained without them, they make the expense of producing a human being similar to that obtained by Farr. For economic purposes we must determine the economic value of man from his earning ability no matter how much he may have cost to produce. The same law of average is a determining factor in this, as in the problem of establishing a life table.

To those who may protest against the determination of the money value of man, Engel quotes from the classical work on anthropometry of Quetelet of France, published in 1885, as follows:

"My investigations and standpoint are so positive that many may shrink from them, some finding them materialistic, others finding in them a desire to extend unduly the limits of the exact sciences and apply mathematics in a region where it does not belong."

Appealing to the advancements made in astronomy in refutation of such ideas and sentiment, Quetelet further says:

"Who will accuse the learned because they have given us in place of the narrow world of the ancients a knowledge of our glorious solar system and have pushed the limits of our starry firmament so far that the mind cannot think of these distances without a religious awe? Surely a knowledge of the wonderful laws that govern the world and have been discovered by learned men makes the power of the divine seem far greater than it did under that conception of the world which a blind superstition would have compelled us to retain."

No. 9, The Evom Table. This word is derived from the first letter of each of the words in the phrase, "economic value of man." It is based upon the method promulgated by Farr, the unit adopted being one dollar a day or three hundred dollars a year. By adopting this unit, computations may be readily made for a person earning any fractional part of one dollar a day, or three hundred dollars a year, or any number of times one dollar a day, or three hundred dollars a year. The rate of discount upon which the computations are made is three and a half per cent. compound interest, per annum.

The earning ability of a person is a composite quantity made up of certain indispensable elements which may be used as factors in a mathematical formula. These are first what nature gives to a man, namely, the structures and functions of the different systems and organs. No one can question the necessity of having these in a fairly healthy state for the normal earning ability of a person. These structures and functions of the different systems and organs of the body may be designated the functional ability of the body, and may be represented by F. as a symbol to be used as a factor in the formula.

The structures and functions of the different systems and organs of the body would have no earning ability if they were not trained to perform certain work successfully. This second indispensable element of the earning ability, the training of the mind and body, is given to man by man. It may be termed the technical ability, and be represented by T. as a symbol to be used as a factor in the formula.

We now come to the third and last indispensable element of the earning ability, namely, the ability a person has from the training of his mind and body to use them successfully in some vocation and receive a remuneration for such services. This third indispensable element of the earning ability may be designated the competing ability, and may be represented by C, as a symbol to be used as a factor in the formula.

For the earning ability, E, may be used as a symbol to represent this composite quantity in the formula and we have:

(1) F x T x C=E, the mathematical formula for the normal earning ability of a person, and for determining damages to the body from injury or disease.

In order to have a practical method for determining damages to the ears, from injury or disease, we must have one that can be used for determining damages to the structure and functions of any other system or organ of the body that may have occurred at the same time. A method that could be used only for one set of organs, like the ears or eyes, and could not be used to ascertain damages to other parts of the body, which may have occurred at the same time, would be of little or no value, because one of the parts of such a method would be purely empirical, and, like the chain which is no stronger than its weakest link, it would be no better than its weakest part. It has been necessary, therefore, for this purpose to devise this mathematical formula for the normal earning ability of the entire body in order that by it we might be able to determine damages to the structures and functions of all the different systems and organs of the entire body, which may occur either singly, or together, at any one time, by one uniform method, which corresponds to that by which the value of any physical force is obtained. It will be seen that it is necessary to treat this subject on the broad general principle of determining damages to any part, or parts, of the body which may have occurred either singly, or together, at any one time.

or a range of 6 of the ten tenths into which the scientific standard of measurement is divided. For persons in vocations requiring lower acuity of sight the range is from 0.05 to 0.5 of the scientific standard of measurement.

After weighing, measuring, comparing and testing all the requirements of the hearing for the average vocations, the conclusion was reached that a range from 0.7 to 0.1 of the scientific standard of measurement is sufficient for the economic standard of measurement. Table 7 has been constructed upon that basis. By this standard of measurement a person may lose 0.3, according to the scientific standard of measurement, and still have hearing sufficient for economic purposes. Again, if he loses 0.9, according to the scientific standard of measurement, he has 0.1 left, but this is not sufficient for economic purposes and that person would then have a total loss of hearing for economic purposes. By this standard we have a range of 0.6 of the scientific for the economic standard which is sufficient for all practical purposes.

For convenience we have conformed partly to the terms employed in the Bureau of Pensions of the United States to designate the different degrees of the loss of hearing on account of their long use and consequent familiarity among medical men. These terms are as follows:

First, slight; second, severe; third, nearly total; and fourth, total, loss of hearing.

By examining Table 7, Standard of Measurement for the Loss of Hearing, it will be observed that it corresponds to Table 6, Standard of Measurement for the Loss of Sight, and also to Table 8, Standard of Measurement for the Loss of Smell, for the Loss of Taste, and for the Loss of Feeling.

With the formulas and tables herewith given, we have a scientific and practical method for determining the economic value of man and the measurement of damages to any part, or parts, of his body, from injury or disease, in a manner equitable to all concerned.

It will be noticed in tables from No. 1 to 4, that T, the technical ability of the mathematical formula for the normal

earning ability of the body, is omitted, because T, the technical ability, is not injured, but limited in proportion to the damages to F, the functional ability of the body, whose impairment includes these damages. T, the technical ability, after it becomes fixed in life remains one, and for this reason also may be discarded, because multiplying any quantity by one does not change its value.

We thus have the mathematical process of the formula to correspond to the simple terms in arithmetic, namely, F, the functional ability of the person, corresponds to the multiplicand, and C, the competing ability of the person, corresponds to the multiplier, while E, the earning ability of the person, corresponds to the product.

The axiom in arithmetic is that increasing the multiplier increases the product, and diminishing the multiplier diminishes the product; that when the divisor is less than one the quotient will be greater than the dividend, and that the reverse of a number is one divided by that number.

In Table 1, the coefficient of C, the competing ability, has been increased (by evolution) to have E, the earning ability, increased whenever it is determined, in a given case of injury, that C, the competing ability, has been damaged to a lesser degree than F, the functional ability.

In Table 2, the coefficient of C, the competing ability, has been diminished (by involution) to have E, the earning ability, decreased whenever it is determined, in a given case of injury, that C, the competing ability, has been damaged to a greater degree than F, the functional ability.

Table 1 represents the reverse process of Table 2, and may be readily understood by the following illustrations: If we produce involution of 1/2 to the 6th power we express it thus, $1/2^6$, and it equals 1/2 x 1/2 x 1/2 x 1/2 x 1/2 x 1/2= 1/64, reduced to a decimal=0.015625. In this example 1/2 is the multiplicand and 1/2 is the multiplier. The product is just as much less than the multiplicand as the multiplier is less than one, namely, it is one-half of it every time it is multiplied. This is the reason why raising a fraction by involution to any given power diminishes its value. This is

what is done in Table 2 to C, the competing ability, in order to have the damage to E, the earning ability, diminished to correspond to the condition of greater damage to C than to F, the functional ability.

In Table 1, the reverse of involution is produced, namely, evolution, and is indicated by the reverse of the number, or one divided by the number, for the index of the exponent, or the root to be sought, thus, $1/64^{1/6}$ or $0.015625^{1/6}=1/2$ or 0.5. If we did not want to increase the value of the fraction to so high a degree we would practice evolution a less number of times as indicated by a smaller number for the denominator of the fractional exponent, or the root to be sought, thus, $1/64^{1/3}$, or $0.015625^{1/3}=1/4$, or 0.25.

$1/64^{1/2}$, or $0.015625^{1/2}=1/8$, or 0.125.

This is what is done in Table 1 to C, the competing ability, in order to have the damage to E, the earning ability, increased to correspond to the condition of lesser degree of damage to C than to F, the functional ability. We have gone into this explanation of the formation of Tables 1, and Tables 2, in order to answer questions that have been asked so many times about them.

In normal conditions the coefficient of F, the functional ability is one, and as C, the competing ability of a person, is composed of the same elements its coefficient is one also, hence E, the earning ability, would be one. When a person has sustained damages to his body, the coefficient of F, and of C, becomes less than one according to the amount of damage sustained. We then have to consider first, whether the damage to F, the functional ability, causes the same degree of damage to C, the competing ability, or second, whether it causes a less degree of damage to C, or third, whether it causes a greater degree of damage to C, than to F, in the vocation of the person.

The treatment of these conditions in the tables is as follows: First, when it is determined that the damage to F, the functional ability, causes the same degree of damage to C, the competing ability, in the vocation followed by the person, then E, the earning ability will be the product of these coefficients. (Table 1, Col. 3.)

Second, when it is determined that the damage to F, the functional ability, causes a lesser degree of damage to C, the competing ability, in the vocation followed by the person, then its coefficient must be increased by evolution before it is multiplied by the coefficient of F. (Table 1, Col. 4, 5, 6, 7, 8 and 9) in order to have E, the earning ability increased to correspond to the lesser degree of damage to C, the competing ability.

Third, when it is determined that the damage to F, the functional ability, causes a greater degree of damage to C, the competing ability, in the vocation followed by the person. then its coefficient must be dimiinished by involution (Table 2, Col. 2, 3, 4, 5, 6, 7, 8, 9 and 10) in order to have E, the earning ability, diminished to correspond to the greater degree of damage to C, the competing ability.

In Table 1, E, the earning ability, after damage to F, the functional ability, from 0.01 to 0.80 is given with C, the competing ability, damaged to the same degree as F, and with C damaged to the tenth degree less than F, and in Table 2, with C damaged to the tenth degree more than F, thus giving sixteen different degrees of damage to C, the competing ability, in the two tables. Table 3 and Table 4 are complementary tables to Table 1 and Table 2, giving the loss on one thousand dollars of the economic value of the person for each of these computations. Thus all the computations are made in an accessible form in these tables and will facilitate the work of solving the problem of determining damages to the body from injury or disease. Table 5 explains itself. It may be used as a standard for comparison in damages for other injuries.

We will now give examples illustrating how readily damages to any part, or parts, of the body may be determined by one uniform method by the use of the formulas and the tables.

We will determine the damages (1) to a person forty-three years old, who is a clerk in a department store, for the total loss of hearing in one ear. He is earning two dollars a day and therefore his gross economic value is in round

numbers $10,000. (No. 9, Evom Table, Col. 2.) The damage to F, the functional ability of the body, according to Table 7, Col. 5, is 0.12. Subtracting this from 1, we have 0.88 for the coefficient of F, and C, which introduced into the formula for the normal earning ability of the body, gives: (5) 0.88 F x 0.88 C=E. If we determine that C, the competing ability of this person, is damaged to the sixth degree less than F, the functional ability, we have: (6) 0.88 F x $0.88C^{1/7}$=E, and we find the value of E, the earning ability, to be 0.8641 (Table 1, Col. 8). If this is the remaining earning ability, we find the loss by subtracting it from 1, and we have 0.1359, the loss on one dollar, or $135.90, the loss of $1,000 (Table 3, Col. 7). As we have just shown that his economic value was $10,000, his economic loss would be ten times $135.90 or $1,359 for the total loss of hearing in one ear. If we add to this the loss in time and expenses incident to the accident, and the loss in suffering in mind and body in consequence of the accident, which was decided to be in this case $336.00, we have a total indemnity of $1,695 for the total loss of hearing in one ear.

We will now determine (2) the damages to a similar clerk for the total loss of hearing in both ears. The damage to F, the functional ability of the body, is twice that for the damage of one ear (Table 7, Col. 5), namely, 0.24. Subtracting this from 1, we have 0.76 for the coefficient of F, and C, which introduced into the formula for the normal earning ability of the body, gives: (7) 0.76 F x 0.76 C=E. It is plainly evident that a person with the total loss of hearing in both ears is damaged as much in C, his competing ability, as in F, his functional ability. If, after due consideration of all the conditions and circumstances in this case, we decide that C, his competing ability, is damaged to the same degree as F, his functional ability, we have: (8) 0.76 F x 0.76 C=E,—Hence E=0.5776 (Table 1, Col. 3), and the loss is 0.4224 on one dollar, or $422.40 on each $1,000 of the economic value of the person (Table 3, Col. 2.) Multiplying this loss by ten, because his economic value was $10,000, we have $4,224. If we add to this loss, the loss in time and his expenses incident to

the accident and the loss in suffering in his mind and body in consequence of the accident, which in this case was decided to be $596, we have the total indemnity of $4,820 for the injury which caused the loss of hearing in both ears. This is $3,125 more indemnity than for the total loss of hearing in one ear.

An indemnity for the loss of any part of the hearing in one ear, or in both ears, as given in Table 7, may be readily worked out in the same way. The question to be determined is the amount of the loss of the hearing in one ear, or in both ears, and the effect this loss has upon C, the competing ability, in the vocation of the person.

We will now show how readily we can determine damages to any part, or parts, of the body, which may have occurred at the same time as the accident which caused the loss of hearing.

We will determine (3) what the indemnity shall be for the total loss of the hearing in one ear, and the total loss of sight of one eye. Both of these losses occur in unit g, factor w, and therefore may be added together (Tables 6, and 7, Col. 5). We find the same to be 0.30. Subtracting this from 1, we have 0.70 for the coefficient of F, and C, and we have: (9) 0.70 F x 0.70 C=E. After due consideration of all the conditions and circumstances of C, the competing ability of this person in his vocation as a clerk in the department store, it is decided that C, his competing ability, is damaged to the sixth degree less than F, the functional ability of the body, and we have: (10) $0.70\ F \times 070C^{1/7}=E$. Hence E, his earning ability, will be 0.6650 (Table 1, Col. 8), and the loss on one dollar is 0.335, or $335 on $1,000 (Table 3, Col. 7). Multiplying this by ten, we have $3,350 as an indemnity for the total loss of hearing in one ear and the total loss of sight of one eye. If we add the loss of his time and expenses and the loss in suffering in his mind and body in consequence of the injury, which was in this case decided to be $324, we have $3,674 as a total indemnity for the total loss of hearing in one ear and the total loss of sight in one eye.

For example (4), the indemnity for the total loss of hearing in both ears and for the total loss of sight in both eyes for a person earning a similar amount would be ascertained as follows: In table 6, and table 7, we find for these four losses 0.60. Subtracting this from 1, we have 0.40 for the coefficient of F, and C, the functional and technical abilities, hence: (11) 0.40 F x 0.40 C=E. The question arises at once, what is the condition of C, the competing ability, in this person, totally blind and deaf? It is plainly evident that he has no competing ability, hence, the exponent of C, becomes O, and we express it in the formula thus: (12) 0.40 F x 0.40C°=Therefore, E=O, because in any computation, when one of the factors becomes O the product is O also. The loss would be total or $10,000. If we add the loss in time and expenses before this indemnity is paid and the loss in suffering in mind and body in consequence of this serious loss of both sight and hearing, which in this case was determined to be $2,500, we have $12,500 for the total indemnity for this person, forty-three years old, earning $2.00 per day. If he were earning $3.00 per day, or $900 a year, the indemnity would be $17,500, assuming the expense for loss of time and suffering in mind and body were the same. If he were earning $4.00 per day, or $1,200 per year, the other expenses being the same, the indemnity would be $22,500 and so on. The only other way of determining damages to the body is by empirical methods, using addition and subtraction. This example shows how absurd this would be, for if the other two factors in unit g were normal, which is a conceivable condition, because this person might be totally blind and totally deaf, and the other two factors, u, the brain, and v, the spinal cord, might be normal, and we obtained the loss by subtraction we would have two factors of this unit left. If the other three units of the body were normal, which is also a conceivable condition, we would have nine other factors, which added to the two of unit g would make 11. Therefore, by subtraction and addition we would have 11/12 of F, the functional ability of the body, left when that person was totally blind and deaf.

We will (5) determine the loss to a person earning a similar amount, who in an accident sustains a total loss of hearing in one ear, the total loss of sight in one eye and the loss of the left hand at the wrist joint. According to Table 5, the loss in unit a is 0.20, and therefore 0.80 is left. According to Table 6, and Table 7, the loss to unit g is 0.30, and therefore 0.70 is left. As these losses occur in two different units their coefficients must be multiplied together to determine F, the remaining functional ability of the body, and we have: (13) 0.80 a x 0.70 g=F. Hence, F= 0.56. If we determine from all the circumstances in this case that C, his competing ability, is damaged to the first degree more than F, his functional ability, we have: (14) 0.56 F x 0.56 C^2=E. Therefore, E= 0.1756 (Table 2, Col. 2), and the loss is 0.8244 on one dollar, or $824.40 on $1,000, and ten times that on $10,000, or $8,244. If we add to this loss, the other two losses incident to the injury, which were decided to be $1,648, we have $9,892 as the total indemnity for the total loss of hearing in one ear, total loss of sight in one eye and the loss of the left hand at the wrist joint, in a person earning two dollars per day, at the age of forty-three.

For example (6), we assume that a clerk, forty-three years old, in a department store, died from an accident that made the corporation liable for the loss of his life. In the five examples given here to illustrate the application of our method, the damage has been adjusted upon the gross economic value. When a person survives an accident the damage must be adjusted upon that basis because it costs that person just as much to live after the accident as before it occurred. When a person does not survive an accident but dies, there are no living expenses incurred for him. Hence the damage must be adjusted upon that basis, which is his net economic value. The net economic value of a person is what he earns less his own personal living expenses. (No. 9, Evom Table). In this case the clerk was earning $2.00 per day, or $600 per year, and his personal living expenses were decided to have been $1.00 a day, or $300 per year. Therefore his net economic value is just half $10,000, his

gross economic value, or $5,000 (No. 9, Evom Table, Col 2). This means that $5,000 put at interest and paid out in the form of an annuity, at 3½ per cent.—the same rate by which his economic value is computed—will last just as long as the clerk was expected to live and earn this amount of money over and above his personal expenses according to the law of average. The expenses incident to the accident and the loss in suffering in mind and body in consequence of the accident were agreed upon as $1,000, which added to $5,000 makes $6,000 as the total indemnity for the corporation to pay for the accident which caused the loss of the life of this clerk, earning $2.00 per day, or $600 per year, at the age of forty-three.

In 1904, I read a paper before the National Association of United States Pension Examining Surgeons and Members of the Bureau of Pensions of the United States at Atlantic City, in which I referred to the fact that the pensions then given were not only based upon empiricism, but were full of inconsistencies and absurdities, and did not do justice to the soldiers and sailors, and therefore should be discarded for scientific methods which would do away with absurdities and do justice to them. The result was that the pensions were revised, and this revision took effect in ten of the principal pensions in 1905, giving a total increase of $1,968 each year.

To illustrate some of the inequalities and absurdities existing in the Bureau of Pensions, reference may be made to the two standards for disabilities. The standard for minor disabilities is anchylosis of the wrist joint, which is rated 8/18, and a pension of $8.00 per month is given. If any disability is considered to be equal to anchylosis of the wrist it is rated the same, and a pension for the same amount is given. If a disability is considered to be any fractional part of 8/18 it is rated accordingly; for instance, nearly total deafness in one ear is thought to be three-fourths of the disability of anchylosis of the wrist and is therefore rated 6/18, and a pension of $6.00 per month is given, while total deafness in one ear is considered to be one and a fourth times as much disability as anchylosis of the wrist, and

therefore, is rated 10/18, and a pension of $10 per month is given. If the pension for nearly total loss of hearing in one ear is increased to correspond with the increase in the pension for total disability from $72 to $100 per month, it should now be $8.32 per month, and for total loss of hearing in one ear it should now be $13.88 per month. If we compare these pensions with those which may be obtained by the scientific methods of physical economics, we find, according to Table 7, Col. 5, the standard of measurement for nearly total loss of hearing of one ear of the 1° is 0.08. Subtracting this from 1 we have 0.92 which becomes the coefficient of F. and C. The other ear being normal, C, the competing ability of the person, would be damaged in most vocations to the least degree, and we have in the formula: (15) $0.92\,F \times 0.92\,C^{1/10} = E$. Hence, $E = 0.9126$ (Table 1, Col. 9), and the loss would be 0.874 for which a pension of $8.74 should be given. The total loss of hearing in one ear has been worked out in one of the examples already given: (5) $0.88\,F \times 0.88\,C^{1/7} = E$. Hence, $E = 0.8641$, and the loss is 0.1359, and a pension of $13.59 should be given when the pension for total disability is $100 per month.

The loss of sight in one eye is considered a disability of one and a half times that of anchylosis of the wrist, and therefore is rated 12/18, and a pension of $12 per month is given, while the loss of an eye is considered two and one-eighth times the disability of anchylosis of the wrist, and therefore is rated 17/18, and a pension of $17 per month is given. If the pensions for total loss of sight in one eye, and for the total loss of an eye, are increased to correspond with the increase in the pensions for total disability from $72 to $100 per month, the former should be increased from $12 to $16.67 per month, and the latter from $17 to $23.61 per month. This is about the same amount as is obtained when the total loss of sight in one eye is determined to affect C, the competing ability of a person, to the least degree, in the vocation he follows as expressed in the formula for the normal earning ability of the body: (16) $0.82\,F \times 0.82\,C^{1/10} = E$. Hence, $E = 0.8036$ (Table 1, Col. 9), and the loss is 0.1964, and a pension

of $19.64 per month should be given for the total loss of sight in one eye. For the total loss of an eye, the damage to C, the competing ability, would be greater, and if in this loss it is determined that C, the competing ability, is damaged to the second degree less than F, the functional ability, in the vocation followed, we have: $0.82\ F \times 0.82\ C^{1/3} = E$. Hence $E = 0.7675$ (Table 1, Col. 5), and the loss is 0.2325, for which loss a pension of $23.25 per month should be given for the total loss of an eye.

Minor disabilities are rated from 2/18 to 17/18, and a pension is given in dollars to correspond with the number of the numerator. Although the pension for total disability was increased from $72 per month to $100 per month, the pensions for minor disabilities have not been increased accordingly.

The other standard is the degree of disability equivalent to the loss of a hand, or a foot, and a pension of $24 per month is given. This method does not provide for a pension to be given for $18, $19, $20, $21, $22 or $23 per month, as if there were no disabilities for which pensions could be given for these amounts. When a disability is considered equivalent to that of the loss of a hand or foot, it is rated the same, and a pension of $24 per month is given. When, however, a soldier or sailor has actually lost a hand at the wrist joint, or a foot at the ankle joint, a pension of $30 per month is given. This difference of $6 per month may have been in recognition of the greater damage to the competing ability of a soldier or sailor when he had actually lost a hand or a foot than when he suffered from a disability equivalent to the loss of a hand or a foot because when a hand or a foot was lost it could be seen by everybody, whereas the equivalent disability might not be so readily seen. When, however, a hand and a foot were lost, a pension of $36 per month was given. In this double disability there was only an increase of $6 per month. This is altogether insufficient and does a great injustice to any soldier or sailor who has sustained such a serious injury, as may be shown from the scientific methods of physical economics. According to Table

5, the standard of measurement for the loss of a hand or a foot is 0.20. Therefore when this loss alone is sustained and it is expressed in the formula for the normal earning ability of the body, we have: (18) 0.80 F x 0.80 C=E. If the loss were a foot, and we determined that C, the competing ability of the person, was damaged to the first degree less than F, the functional ability, in the vocation followed, then the same would be expressed in the formula for the normal earning ability of the body as follows: (19) 0.80 F x 0.80 $C^{1/2}$ =E. Hence, E=0.7152 (Table 1, Col. 4). The loss would be 0.2848, and, therefore, a pension of $28.48 per month should be given for the loss of a foot when a pension of $100 per month is given for total disability. If the left hand were lost, in a right-handed person, and we determined that C, the competing ability, was damaged to the same degree as F, the functional ability, then it would be expressed in the formula for the normal earning ability of the body as follows: (20) 0.80 F x 0.80 C=E. Hence, E=0.64 (Table 1, Col. 3), and the loss would be 0.36. Therefore, a pension of $36 a month should be given for the loss of the left hand. If the right hand were lost in a right handed person, and we determined that C, the competing ability of the person, was damaged to the first degree more than F, the functional ability, in the vocation followed, then it would be expressed in the formula for the normal earning ability of the body as follows: (21) 0.80 F x 0.80 C^2 =E. Hence, E=0.512 (Table 2, Col. 2) and the loss would be 0.4880, and a pension of $48.80 per month should be given for the loss of the right hand. If a hand and a foot were lost we find the sum of these two losses in Table 5 to be 0.40, and subtracting this from 1, we have in the formula for the normal earning ability of the body: (22) 0.60 F x 0.60 C=E. We must then determine how this serious loss affects the person in the vocation followed. It is self evident that the loss of a hand and a foot at the same time would damage C, the competing ability of a person, more than the loss of either one alone. We then determined that the damage to C, the competing ability, was equal to the first degree less than to F, the

functional ability, when a foot was lost at the ankle joint, that the former was damaged to the same degree when the left hand was lost, and that the former was damaged to the first degree more than the latter when the right hand was lost. It would be conservative then to decide that when a foot and left hand were lost, C, the competing ability, would be damaged to the same degree as F, the functional ability, and we have: (23) 0.60 F x 0.60 C=E. Hence, E=0.36 (Table 1, Col. 3), and the loss would be 0.64, for which a pension of $64 per month should be given for the loss of a foot and the left hand. If the right hand and a foot were lost it would be conservative to determine that C, the competing ability, was damaged to the first degree more than F, the functional ability, because when the right hand alone was lost, we determined that C, the competing ability, was damaged to the first degree more than F, the functional ability, and we have: (24) 0.60 F x 0.60 C^2=E. Hence, E=0.216 (Table 2, Col. 2) and the loss would be 0.784, for which a pension of $78.40 should be given for the loss of a foot and the right hand.

In the revision of the pensions brought about by my paper the pension for the loss of a hand and a foot was increased from $36 to $60 per month. That C, the competing ability, is still not duly recognized in the Bureau of Pensions is very evident from the cases just cited of the loss of a foot, a left hand, or a right hand, or when a foot is lost with the right or left hand. The pension for the loss of both hands was established by a vote of Congress to be $100 per month, whereas the pension for total disability, like that of the loss of both eyes, was $72 per month. The pension for total loss of hearing in both ears was $30 per month. This was increased in the revision of the pensions to $40 per month. The latter is very nearly the same as is obtained in equation (8), 0.74 F x 0.76 C=E, when E=0.5776 (Table 1, Col. 3) and the damage is 0.4224, or $42.24 per month when the pension for total disability is $100 per month.

These examples might be multiplied indefinitely, but a sufficient number and variety have been given to show how damages to any part, or parts, of the body may be obtained by

the formulas and the scientific and economic standards of measurement.

Those who have considered this subject but little often express distrust of any method which employs a mathematical formula that determines E, the earning ability of a person, and his economic value therefrom, in dollars and cents. They say there is so much uncertainty about C, the competing ability of persons, that they do not see how it can be determined with any degree of reliability. When, however, they admit that life itself is the most uncertain thing in this world, and from its existence the law of average is deduced, upon which the life table is constructed, and life insurance, the largest business of the world, is projected and carried on successfully, they become convinced that it is feasible and practical, because they then realize that the principal factor in determining E, the earning ability of a person, and his economic value, depends upon the same law of average as that by which the life table itself is constructed.

There may be a difference of opinion as to the standards of measurement for determining the loss to F, the functional ability of the body, in any given case of damage. The tables here given may not be the final determination in these problems, but they are the best we have to offer after studying the subject for years, and testing them with all the conditions and circumstances connected with such damages, and the results obtained by empirical methods in courts of law and in the Bureau of Pensions of the United States. At first sight the amount given in the tables for the loss specified would seem to be too low, but it must be remembered that the standard of measurement for the functional loss of any part, or parts, of the body must be so low that the loss to F, the functional ability of the body, would be equal to this amount to a person in any vocation. In determining these standards of measurement, for the partial and total loss of hearing, this fact, that they must be made so low as to meet this loss equitably in persons of every vocation, has been kept constantly in view. We feel sure then that

when a person has sustained a total or partial loss of hearing in one ear or both ears, and the damage to F, the functional ability of the body, has been adjusted by this table, the loss to E, the earning ability, can always be equitably determined in every case by considering how this damage affects C, the competing ability, in the vocation the person follows. Hence, we are forced to the conclusion that the standard of measurement determined upon in Table 7 meets and satisfies every condition connected with the partial or complete loss of hearing in one or both ears for determining damages to E, the earning ability of a person, in a manner equitable to all concerned.

TABLE I

(1)	(2)		(3)	(4)	(5)	(6)	(7)	(8)	(9)
Loss in F	Computations made when C, the competing ability, is damaged to the same degree as F, the functional ability, and when C, the competing ability, is damaged to six different degrees less than F, the functional ability								
	F	C	E=	$C^{1.2}$ E=	$C^{1.3}$ E=	$C^{1.4}$ E=	$C^{1.5}$ E=	$C^{1.7}$ E=	$C^{1.10}$ E=
0.01	.99	.99	=0.9801	0.985	0.9865	0.9875	0.988	0.9884	0.989
0.02	.98	.98	=0.9604	0.9702	0.9731	0.9751	0.976	0.9770	0.9780
0.03	.97	.97	=0.9409	0.9554	0.9593	0.9622	0.9641	0.9651	0.9670
0.04	.96	.96	=0.9216	0.9408	0.9465	0.9504	0.9523	0.9542	0.9561
0.05	.95	.95	=0.9025	0.9262	0.9388	0.9376	0.9405	0.9424	0.9452
0.06	.94	.94	=0.8836	0.9118	0.9202	0.9259	0.9277	0.9315	0.9343
0.07	.93	.93	=0.8649	0.8965	0.9067	0.9132	0.9151	0.9197	0.9234
0.08	.92	.92	=0.8464	0.8822	0.8942	0.9006	0.9043	0.9089	0.9126
0.09	.91	.91	=0.8281	0.8681	0.8898	0.8881	0.8927	0.8972	0.9001
0.10	.90	.90	=0.81	0.8541	0.8685	0.8766	0.8811	0.8865	0.8901
0.11	.89	.89	=0.7921	0.8392	0.8552	0.8641	0.8695	0.8748	0.8784
0.12	.88	.88	=0.7744	0.8254	0.8430	0.8527	0.8580	0.8641	0.8685
0.13	.87	.87	=0.7569	0.8108	0.8308	0.8404	0.8456	0.8526	0.8578
0.14	.86	.86	=0.7396	0.7972	0.8187	0.8290	0.8342	0.8419	0.8571
0.15	.85	.85	=0.7225	0.7828	0.8058	0.8160	0.8228	0.8304	0.8347
0.16	.84	.84	=0.7056	0.7694	0.7926	0.8047	0.8114	0.8190	0.8257
0.17	.83	.83	=0.6889	0.7561	0.7802	0.7885	0.7992	0.8075	0.8142
0.18	.82	.82	=0.6724	0.7429	0.7675	0.7806	0.7880	0.7970	0.8036
0.19	.81	.81	=0.6561	0.729	0.7549	0.7686	0.7759	0.7857	0.7929
0.20	.80	.80	=0.64	0.7152	0.7424	0.7568	0.7648	0.7752	0.7824
0.21	.79	.79	=0.6241	0.6915	0.7299	0.7449	0.7528	0.7639	0.7710
0.22	.78	.78	=0.6084	0.6887	0.7183	0.7332	0.7417	0.7527	0.7605
0.23	.77	.77	=0.5929	0.6752	0.7060	0.7214	0.7299	0.7415	0.7499
0.24	.76	.76	=0.5776	0.6627	0.6938	0.7098	0.7189	0.7303	0.7394
0.25	.75	.75	=0.5625	0.6495	0.6817	0.6982	0.7072	0.7192	0.7282
0.26	.74	.74	=0.5476	0.6371	0.6697	0.6867	0.6963	0.7089	0.7178
0.27	.73	.73	=0.5329	0.6241	0.6577	0.6752	0.6847	0.6978	0.7073
0.28	.72	.72	=0.5184	0.6112	0.6458	0.6638	0.6739	0.6868	0.6969
0.29	.71	.71	=0.5041	0.5985	0.6340	0.6517	0.6624	0.6759	0.6858
0.30	.70	.70	=0.49	0.5859	0.6223	0.6405	0.6517	0.6650	0.6755
0.31	.69	.69	=0.4761	0.5738	0.6099	0.6285	0.6403	0.6541	0.6679
0.32	.68	.68	=0.4624	0.561	0.5984	0.6174	0.6296	0.6432	0.6541
0.33	.67	.67	=0.4489	0.5487	0.5862	0.6056	0.6184	0.6324	0.6432
0.34	.66	.66	=0.4356	0.5365	0.5748	0.5946	0.6078	0.6217	0.6329
0.35	.65	.65	=0.4225	0.5239	0.5629	0.5830	0.5967	0.6110	0.6220
0.36	.64	.64	=0.4096	0.512	0.5516	0.5721	0.5856	0.6003	0.6118
0.37	.63	.63	=0.3969	0.4995	0.5399	0.5607	0.5745	0.5896	0.6010
0.38	.62	.62	=0.3844	0.4879	0.5288	0.5499	0.5635	0.5790	0.5908
0.39	.61	.61	=0.3721	0.4758	0.5172	0.5386	0.5526	0.5685	0.5801
0.40	.60	.60	=0.36	0.4644	0.5058	0.5280	0.5418	0.5580	0.5700
0.41	.59	.59	=0.3481	0.4525	0.4944	0.5174	0.531	0.5475	0.5593
0.42	.58	.58	=0.3364	0.4413	0.4837	0.5075	0.5202	0.5370	0.5492
0.43	.57	.57	=0.3249	0.4297	0.4725	0.4959	0.5090	0.5261	0.5386
0.44	.56	.56	=0.3136	0.4188	0.4614	0.4884	0.4984	0.5157	0.5286
0.45	.55	.55	=0.3025	0.4075	0.4504	0.4735	0.4878	0.5049	0.5181
0.46	.54	.54	=0.2916	0.3969	0.4395	0.4627	0.4773	0.4946	0.5076
0.47	.53	.53	=0.2809	0.3858	0.4287	0.4520	0.4664	0.4838	0.4971
0.48	.52	.52	=0.2704	0.3749	0.4180	0.4414	0.4560	0.4737	0.4872
0.49	.51	.51	=0.2601	0.3641	0.4080	0.4309	0.4447	0.4630	0.4763
0.50	.50	.50	=0.25	0.3535	0.3985	0.4205	0.4350	0.4530	0.4665
0.51	.49	.49	=0.2401	0.3430	0.3871	0.4096	0.4243	0.4424	0.4561

TABLE I—Continued

(1)	(2)		(3)	(4)	(5)	(6)	(7)	(8)	(9)
Loss in F	Computations made when C, the competing ability, is damaged to the same degree as F, the functional ability, and when C, the competing ability, is damaged to six different degrees less than F, the functional ability								
	F	C	E=	$C^{1.2}$ E=	$C^{1.3}$ E=	$C^{1.4}$ E=	$C^{1.5}$ E=	$C^{1.7}$ E=	$C^{1.10}$E=
0.52	.48	.48	=0.2304	0.3326	0.3758	0.3993	0.4142	0.4324	0.4459
0.53	.47	.47	=0.2209	0.3219	0.3651	0.3886	0.4037	0.4220	0.4356
0.54	.46	.46	=0.2116	0.3118	0.3551	0.3785	0.3937	0.4117	0.4255
0.55	.45	.45	=0.2025	0.3015	0.3447	0.3681	0.3834	0.4014	0.4153
0.56	.44	.44	=0.1936	0.2917	0.3348	0.3581	0.3735	0.3911	0.4052
0.57	.43	.43	=0.1849	0.2795	0.3225	0.3478	0.3633	0.3809	0.3951
0.58	.42	.42	=0.1764	0.2721	0.3145	0.3381	0.3532	0.3708	0.3851
0.59	.41	.41	=0.1681	0.2624	0.3046	0.3321	0.3431	0.3595	0.3747
0.60	.40	.40	=0.16	0.2528	0.2948	0.3180	0.3332	0.3504	0.3648
0.61	.39	.39	=0.1521	0.2433	0.2847	0.3081	0.3229	0.3408	0.3549
0.62	.38	.38	=0.1444	0.2340	0.2751	0.2983	0.3131	0.3309	0.3450
0.63	.37	.37	=0.1369	0.2249	0.2652	0.2886	0.3030	0.3207	0.3348
0.64	.36	.36	=0.1296	0.216	0.2559	0.2791	0.2934	0.3110	0.3250
0.65	.35	.35	=0.1225	0.2068	0.2481	0.2709	0.2835	0.3010	0.3150
0.66	.34	.34	=0.1156	0.1982	0.2373	0.2597	0.2740	0.2913	0.3053
0.67	.33	.33	=0.1089	0.1894	0.2280	0.2501	0.2643	0.2814	0.2953
0.68	.32	.32	=0.1024	0.1811	0.2188	0.2406	0.2547	0.2720	0.2854
0.69	.31	.31	=0.0961	0.1726	0.2095	0.2312	0.2452	0.2622	0.2755
0.70	.30	.30	=0.09	0.1644	0.2007	0.222	0.2358	0.2526	0.2658
0.71	.29	.29	=0.0841	0.1560	0.1902	0.2125	0.2262	0.2430	0.2560
0.72	.28	.28	=0.0784	0.1481	0.1831	0.2035	0.2170	0.2335	0.2464
0.73	.27	.27	=0.0729	0.1401	0.1744	0.1944	0.2076	0.2238	0.2367
0.74	.26	.26	=0.0676	0.1326	0.1658	0.1856	0.1986	0.2145	0.2272
0.75	.25	.25	=0.0625	0.1250	0.1572	0.1767	0.1895	0.2050	0.2175
0.76	.24	.24	=0.0576	0.1176	0.1490	0.1680	0.1804	0.1958	0.2080
0.77	.23	.23	=0.0529	0.1101	0.1407	0.1591	0.1713	0.1865	0.1984
0.78	.22	.22	=0.0484	0.1031	0.1328	0.1507	0.1625	0.1773	0.1889
0.79	.21	.21	=0.0441	0.0961	0.1247	0.1421	0.1537	0.168	0.1795
0.80	.20	.20	=0.04	0.0894	0.1170	0.1338	0.1450	0.1588	0.1702

TABLE 2

(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)
Loss in F	Computations made when C, the competing ability, is damaged from 2 to 10 degrees more than F, the functional ability								
	$C^2E=$	$C^3E=$	$C^4E=$	$C^5E=$	$C^6E=$	$C^7E=$	$C^8E=$	$C^9E=$	$C^{10}E=$
0.01	0.9702	0.9604	0.9507	0.9411	0.9316	0.9222	09129	0.9039	0.8948
0.02	0.9415	0.9222	0.9037	0.8856	0.8678	0.8504	0.8333	0.8166	0.8002
0.03	0.9126	0.8852	0.8586	0.8328	0.8078	0.7835	0.7599	0.7371	0.7149
0.04	0.8847	0.8493	0.8153	0.7826	0.7512	0.7211	0.6922	0.6645	0.6379
0.05	0.8573	0.8144	0.7736	0.7349	0.6981	0.6631	0.6299	0.5984	0.5684
0.06	0.8305	0.7806	0.7337	0.6896	0.6482	0.6093	0.5727	0.5383	0.5060
0.07	0.8043	0.7479	0.6955	0.6468	0.6015	0.5593	0.5251	0.4883	0.4541
0.08	0.7786	0.7163	0.6589	0.6061	0.5515	0.5073	0.4667	0.4293	0.3949
0.09	0.7535	0.6856	0.6238	0.5676	0.5165	0.4700	0.4277	0.3692	0.3359
0.10	0.729	0.6561	0.5904	0.5313	0.4781	0.4302	0.3871	0.3483	0.3133
0.11	0.7049	0.6273	0.5582	0.4967	0.4420	0.3933	0.3500	0.3115	0.2772
0.12	0.6814	0.5996	0.5276	0.4642	0.4084	0.3593	0.3161	0.2781	0.2447
0.13	0.6583	0.5727	0.4982	0.4334	0.3770	0.3279	0.2852	0.2481	0.2158
0.14	0.6360	0.5469	0.4703	0.4044	0.3477	0.293	0.2519	0.2166	0.1862
0.15	0.6141	0.5219	0.4436	0.377	0.3204	0.2723	0.2314	0.1966	0.1671
0.16	0.5927	0.4978	0.4181	0.3512	0.295	0.2478	0.2081	0.1748	0.1467
0.17	0.5717	0.4745	0.3938	0.3268	0.2712	0.225	0.1887	0.1566	0.1299
0.18	0.5513	0.4520	0.3706	0.3038	0.2491	0.2042	0.1674	0.1372	0.1125
0.19	0.5314	0.4304	0.3486	0.2823	0.2286	0.1851	0.1499	0.1214	0.0983
0.20	0.512	0.4096	0.3276	0.2620	0.2096	0.1676	0.134	0.1072	0.0859
0.21	0.4930	0.3894	0.3076	0.2430	0.1919	0.1516	0.1197	0.0945	0.0746
0.22	0.4745	0.3701	0.2886	0.2251	0.1755	0.1368	0.1067	0.0832	0.0648
0.23	0.4565	0.3515	0.2706	0.2083	0.1603	0.1234	0.095	0.0731	0.0562
0.24	0.4389	0.3335	0.2534	0.1925	0.1463	0.1111	0.0844	0.0641	0.0487
0.25	0.4218	0.3163	0.2372	0.1779	0.1334	0.1000	0.075	0.0562	0.0421
0.26	0.4052	0.2998	0.2218	0.1641	0.1214	0.0898	0.0664	0.0491	0.0363
0.27	0.3890	0.2839	0.2072	0.1512	0.1103	0.0805	0.0587	0.0428	0.0312
0.28	0.3732	0.2687	0.1934	0.1392	0.1002	0.0721	0.0519	0.0373	0.0268
0.29	0.3579	0.2541	0.1804	0.1280	0.0908	0.0644	0.0457	0.0324	0.023
0.30	0.343	0.2401	0.168	0.1176	0.0823	0.0576	0.0403	0.0282	0.0197
0.31	0.3285	0.2266	0.1563	0.1078	0.0743	0.0512	0.0353	0.0243	0.0167
0.32	0.3144	0.2137	0.1453	0.0988	0.0671	0.0456	0.031	0.0210	0.0142
0.33	0.3007	0.2014	0.1349	0.0903	0.0605	0.0405	0.0271	0.0181	0.0121
0.34	0.2874	0.1896	0.1251	0.0825	0.0544	0.0359	0.0236	0.0155	0.0102
0.35	0.2746	0.1784	0.1159	0.0753	0.0489	0.0317	0.0206	0.0133	0.0086
0.36	0.2621	0.1679	0.1073	0.0686	0.0439	0.028	0.0179	0.0114	0.0072
0.37	0.2500	0.1575	0.0992	0.0624	0.0393	0.0247	0.0155	0.0097	0.0061
0.38	0.2383	0.1477	0.0915	0.0567	0.0351	0.0217	0.0134	0.0083	0.0051
0.39	0.2269	0.1384	0.0844	0.0514	0.0313	0.019	0.0115	0.007	0.0042
0.40	0.216	0.1296	0.0777	0.0466	0.0279	0.0167	0.01	0.006	0.0036
0.41	0.2053	0.1211	0.0714	0.0421	0.0248	0.0146	0.0086	0.005	0.0029
0.42	0.1951	0.1131	0.0655	0.0379	0.0219	0.0127	0.0073	0.0042	0.0024
0.43	0.1851	0.1055	0.0601	0.0342	0.0194	0.011	0.0062	0.0035	0.0019
0.44	0.1756	0.0983	0.055	0.0308	0.0172	0.0096	0.0053	0.0029	0.0016
0.45	0.1663	0.0914	0.0502	0.0275	0.0151	0.0083	0.0045	0.0026	0.0014
0.46	0.1574	0.0849	0.0458	0.0247	0.0133	0.0071	0.0038	0.002	0.001
0.47	0.1488	0.0788	0.0417	0.0221	0.0117	0.0062	0.0032	0.0016	0.0008
0.48	0.1406	0.0731	0.038	0.0197	0.0102	0.0053	0.0027	0.0014	0.0007
0.49	0.1326	0.0676	0.0344	0.0175	0.0089	0.0045	0.0022	0.0011	0.0005
0.50	0.1250	0.0625	0.0312	0.0156	0.0078	0.0039	0.0019	0.0009	0.0004
0.51	0.1176	0.0576	0.0282	0.0138	0.0067	0.0032	0.0015	0.0007	0.0003
0.52	0.1105	0.0530	0.0254	0.0121	0.0058	0.0027	0.0012	0.0005	0.0002
0.53	0.1038	0.0487	0.0228	0.0107	0.0050	0.0023	0.0010	0.0004	0.0001

TABLE 2—Continued

(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)
Loss in F	Computations made when C, the competing ability, is damaged from 2 to 10 degrees more than F, the functional ability								
	C^2 E=	C^3 E=	C^4 E=	C^5 E=	C^6 E=	C^7 E=	C^8 E=	C^9 E=	C^{10} E=
0.54	0.0973	0.0447	0.0205	0.0094	0.0043	0.0019	0.0008	0.0003	0.0001
0.55	0.0911	0.0409	0.0184	0.0082	0.0036	0.0016	0.0007	0.0003	0.0001
0.56	0.0851	0.0374	0.0164	0.0072	0.0031	0.0013	0.0005	0.0002	0.0000
0.57	0.0795	0.0341	0.0146	0.0062	0.0026	0.0011	0.0004	0.0001	
0.58	0.0740	0.0310	0.013	0.0054	0.0022	0.0009	0.0003	0.0001	
0.59	0.0689	0.0282	0.0115	0.0047	0.0019	0.0007	0.0002	0.0000	
0.60	0.064	0.0256	0.0102	0.004	0.0016	0.0006	0.0002		
0.61	0.0593	0.0231	0.009	0.0035	0.0013	0.0005	0.0001		
0.62	0.0548	0.0208	0.0079	0.0030	0.0011	0.0004	0.0001		
0.63	0.0506	0.0187	0.0069	0.0025	0.0009	0.0003	0.0001		
0.64	0.0466	0.0167	0.006	0.0021	0.0007	0.0002	0.0000		
0.65	0.0428	0.0149	0.0052	0.0018	0.0006	0.0001			
0.66	0.0393	0.0133	0.0045	0.0015	0.0005	0.0001			
0.67	0.0359	0.0118	0.0038	0.0012	0.0003	0.0000			
0.68	0.0327	0.0104	0.0033	0.0010	0.0003				
0.69	0.0297	0.0092	0.0028	0.0008	0.0002				
0.70	0.027	0.0081	0.0024	0.0007	0.0002				
0.71	0.0243	0.007	0.002	0.0005	0.0001				
0.72	0.0219	0.0061	0.0017	0.0004	0.0001				
0.73	0.0196	0.0052	0.0014	0.0003	0.0000				
0.74	0.0175	0.0045	0.0011	0.0002					
0.75	0.0156	0.0039	0.0009	0.0002					
0.76	0.0138	0.0032	0.0007	0.0001					
0.77	0.0121	0.0027	0.0005	0.0001					
0.78	0.0106	0.0023	0.0005	0.0001					
0.79	0.0092	0.0019	0.0003	0.0000					
0.80	0.008	0.0016	0.0003						

TABLE 3

(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
Loss in F	Loss on $1,000, when C, the competing ability, is damaged to the same degree as F, the functional ability and when C, the competing ability, is damaged to six different degrees less than F, the functional ability						
	C^{1}	$C^{1.2}$	$C^{1.3}$	$C^{1.4}$	$C^{1.5}$	$C^{1.7}$	$C^{1.10}$
0.01	$ 19.90	$ 15.00	$ 13.50	$ 12.50	$ 12.00	$ 11.60	$ 11.00
0.02	39.60	29.80	26.90	24.90	24.00	23.00	22.00
0.03	59.10	44.60	40.70	37.80	35.90	34.90	33.00
0.04	70.40	59.20	53.50	49.60	47.70	45.80	43.90
0.05	97.50	73.80	66.20	62.40	59.50	57.60	54.80
0.06	116.40	88.20	79.80	74.10	72.30	68.50	65.70
0.07	135.10	103.50	93.30	86.80	84.90	80.30	76.60
0.08	153.60	117.80	105.80	99.40	95.70	91.10	87.40
0.09	171.90	131.90	110.20	111.90	107.30	102.80	99.90
0.10	190.00	145.90	131.50	123.40	118.90	113.50	109.90
0.11	207.90	160.80	144.80	135.90	130.50	125.50	121.60
0.12	225.60	174.60	157.00	147.30	142.00	135.90	131.50
0.13	243.10	189.20	169.20	159.60	154.40	147.40	142.20
0.14	260.40	202.80	181.30	171.00	165.80	158.10	152.90
0.15	277.50	217.20	194.20	184.00	177.20	169.60	165.30
0.16	294.40	230.60	207.40	195.30	188.60	181.00	174.30
0.17	311.10	243.90	219.80	211.50	200.80	192.50	185.80
0.18	327.60	257.10	232.50	219.40	212.00	203.00	196.40
0.19	343.90	271.00	245.10	231.40	224.10	214.30	207.10
0.20	360.00	284.80	257.60	243.20	235.20	224.80	217.60
0.21	375.90	308.50	270.10	255.10	247.20	236.10	229.00
0.22	391.60	311.30	281.70	266.80	258.30	247.30	239.50
0.23	407.10	324.80	294.00	278.60	270.10	258.50	250.10
0.24	422.40	347.30	306.20	290.20	281.10	269.70	260.60
0.25	437.50	350.50	318.30	301.80	292.80	280.80	271.80
0.26	452.40	362.90	330.30	312.30	303.70	291.10	282.20
0.27	467.10	375.90	342.30	324.80	315.30	302.20	292.70
0.28	481.60	388.80	354.20	336.20	326.10	313.20	303.10
0.29	495.90	401.50	366.00	348.30	337.60	324.10	313.50
0.30	510.00	414.10	377.70	359.50	348.30	335.00	324.50
0.31	523.90	426.70	390.10	371.50	359.70	345.90	334.90
0.32	537.60	439.00	401.60	382.60	370.40	356.80	345.90
0.33	551.10	451.30	413.80	394.40	381.60	367.60	356.80
0.34	564.40	463.50	425.20	405.40	392.20	378.30	367.10
0.35	577.50	476.10	437.10	417.00	403.30	389.00	378.00
0.36	590.40	488.00	448.40	427.90	414.40	397.70	388.20
0.37	603.10	500.50	460.10	439.30	425.50	410.40	399.00
0.38	615.60	512.10	471.20	450.10	436.50	421.00	409.20
0.39	627.90	524.20	482.80	461.40	447.40	431.50	419.90
0.40	640.00	535.60	494.20	472.00	458.20	442.00	430.00
0.41	651.90	547.50	505.60	482.60	469.00	452.50	440.70
0.42	663.60	558.70	516.30	492.50	478.80	463.00	450.80
0.43	675.10	570.30	527.50	504.10	491.00	473.90	461.40
0.44	686.40	581.20	538.60	511.60	501.60	484.30	471.40
0.45	697.50	592.50	549.60	526.50	512.20	495.10	481.90
0.46	708.40	603.10	560.50	537.30	522.70	505.40	492.40
0.47	719.10	614.20	571.30	548.00	533.60	516.20	502.90
0.48	729.60	625.10	582.00	558.00	544.00	526.30	512.80
0.49	739.90	635.90	592.00	569.10	555.30	537.00	523.20
0.50	750.00	646.50	601.50	579.50	565.00	547.00	533.50
0.51	759.60	657.00	612.90	590.40	575.70	557.60	543.90

TABLE 3—Continued

(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
Loss in F	Loss on $1,000, when C, the competing ability, is damaged to the same degree as F, the functional ability, and when C, the competing ability, is damaged to six different degrees less than F, the functional ability						
	C^1	C^{1-2}	C^{1-3}	C^{1-4}	C^{1-5}	C^{1-7}	C^{1-10}
0.52	769.60	667.40	624.20	600.70	585.80	567.60	554.10
0.53	779.10	678.10	634.90	611.40	596.30	578.00	564.40
0.54	788.40	688.20	644.90	621.50	606.40	588.30	574.50
0.55	795.50	698.50	655.30	631.90	616.60	598.60	584.70
0.56	806.40	708.30	665.20	641.90	626.50	608.90	594.80
0.57	815.10	720.50	677.50	652.20	637.70	619.10	604.90
0.58	823.50	727.90	685.50	661.90	646.80	629.20	614.90
0.59	831.90	737.60	695.40	667.90	656.90	640.90	625.30
0.60	840.00	747.20	705.20	682.00	666.80	649.20	635.20
0.61	847.10	756.70	715.30	691.90	677.10	659.20	645.10
0.62	855.60	766.00	724.90	701.70	686.90	669.10	655.00
0.63	863.10	775.10	734.80	711.40	697.00	679.30	665.20
0.64	870.40	784.00	744.10	720.90	706.60	689.00	675.00
0.65	877.50	793.20	751.90	729.10	716.50	699.00	685.00
0.66	884.40	801.80	762.70	740.30	726.00	708.70	694.70
0.67	891.10	810.60	772.00	749.90	735.70	718.60	704.70
0.68	897.60	818.90	781.20	759.40	745.30	728.00	714.60
0.69	903.90	827.40	790.50	768.80	754.80	737.80	724.50
0.70	910.00	835.60	799.30	778.00	764.20	747.40	734.20
0.71	915.90	844.00	809.80	787.50	773.80	757.00	744.00
0.72	962.60	851.90	816.90	796.50	783.00	766.50	753.60
0.73	927.10	858.90	825.60	805.60	792.40	776.20	763.30
0.74	932.40	867.40	834.20	814.40	801.40	785.50	772.80
0.75	937.50	875.00	842.80	823.30	810.50	795.00	782,50
0.76	942.40	882.40	851.00	832.00	819.60	804.20	792.00
0.77	947.10	889.90	859.30	840.90	828.70	813.50	801.60
0.78	951.50	896.90	867.20	849.30	837.50	822.70	811.10
0.79	955.10	903.90	875.30	857.90	846.30	832.00	820.50
0.80	960.00	910.60	883.00	866.20	855.00	841.20	829.80

TABLE 4

(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)
Loss in F	Loss on $1,000 when C, the competing ability, is damaged from 2 to 10 degrees more than F, the functional ability								
	C^2	C^3	C^4	C^5	C^6	C^7	C^8	C^9	C^{10}
0.01	$ 29.80	$ 39.60	$ 49.30	$ 58.90	$ 68.40	$ 77.80	$ 87.10	$ 96.10	$105.20
0.02	58.50	97.80	96.30	114.40	132.20	149.60	166.70	183.40	199.80
0.03	87.40	114.80	141.40	167.20	192.20	216.50	240.10	262.90	285.10
0.04	115.30	150.70	184.70	217.40	248.80	278.90	307.80	335.50	362.10
0.05	142.70	185.60	226.40	265.10	301.90	336.90	370.10	401.60	431.60
0.06	169.50	219.40	266.30	310.40	351.80	390.70	427.30	461.70	494.00
0.07	195.70	252.10	304.50	353.20	398.50	440.70	474.90	511.70	545.90
0.08	221.40	283.70	341.10	393.90	448.50	492.70	533.30	570.70	605.10
0.09	246.50	314.40	376.20	432.40	483.50	530.00	572.30	630.80	664.10
0.10	271.00	343.90	409.60	468.70	521.90	569.80	612.90	651.70	686.70
0.11	295.10	372.70	441.80	503.80	558.00	606.70	650.00	688.50	722.80
0.12	318.60	400.40	472.40	535.80	591.60	640.70	683.90	721.90	755.30
0.13	341.70	427.30	501.80	566.60	623.00	672.10	714.80	751.90	784.20
0.14	364.00	453.10	529.70	595.60	652.30	707.00	748.10	783.40	813.80
0.15	385.90	478.10	556.40	623.00	679.60	727.70	768.60	803.40	832.90
0.16	407.30	502.20	581.90	648.80	705.00	752.20	791.90	825.20	853.30
0.17	428.30	525.50	606.20	673.20	728.80	775.00	811.30	843.40	870.10
0.18	448.70	548.00	629.40	696.20	750.90	795.80	832.60	862.80	887.50
0.19	468.60	569.60	651.40	717.70	771.40	814.90	850.10	878.60	901.70
0.20	488.00	590.40	672.40	738.00	790.40	832.40	866.00	892.80	914.10
0.21	507.00	610.60	692.40	757.00	808.10	848.40	880.30	905.50	925.40
0.22	525.50	629.90	711.40	774.90	824.50	863.20	893.30	916.80	935.20
0.23	543.50	648.50	729.40	791.70	839.70	876.60	905.00	926.90	943.80
0.24	561.10	666.50	746.60	807.50	853.70	888.90	915.60	935.90	951.30
0.25	578.20	683.70	762.80	822.10	866.60	900.00	925.00	943.80	959.90
0.26	594.80	700.20	778.20	835.90	878.60	910.20	933.60	950.90	963.70
0.27	611.00	716.10	792.80	848.80	889.70	919.50	941.30	957.20	968.80
0.28	626.80	731.30	806.60	860.80	899.80	927.90	948.10	962.70	973.20
0.29	642.10	745.90	819.60	872.00	909.20	935.60	954.30	967.60	977.00
0.30	657.00	759.90	832.00	882.40	917.70	942.40	959.70	971.80	980.30
0.31	671.50	773.40	843.70	892.20	925.70	948.80	964.70	975.70	983.30
0.32	685.60	786.30	854.70	901.20	932.90	954.40	969.00	979.00	985.80
0.33	699.30	798.60	865.10	907.70	939.50	959.50	972.90	981.90	987.90
0.34	712.60	810.40	874.90	917.50	945.60	964.10	976.40	984.50	989.80
0.35	725.40	821.60	884.10	924.70	951.10	968.30	979.40	986.70	991.40
0.36	737.90	832.10	892.70	931.40	956.10	972.00	982.10	988.60	992.80
0.37	750.00	842.50	900.80	937.60	960.70	975.30	984.50	990.30	993.90
0.38	761.70	852.30	908.50	943.30	964.90	978.30	986.60	991.70	994.90
0.39	773.10	861.60	915.60	948.60	968.70	981.00	988.50	993.00	995.80
0.40	784.00	870.40	922.30	953.40	972.10	983.30	990.00	994.00	996.40
0.41	794.70	878.90	928.60	957.90	975.20	985.40	991.40	995.00	997.10
0.42	804.90	886.90	934.50	962.10	978.10	987.30	992.70	995.80	997.60
0.43	814.90	894.50	939.90	965.80	980.60	989.00	993.80	996.50	998.10
0.44	824.40	901.70	945.00	969.20	982.80	990.40	994.70	997.10	998.40
0.45	833.70	908.60	949.80	972.50	984.90	991.70	995.50	997.40	998.60
0.46	824.60	915.10	954.20	975.30	986.70	992.90	996.20	998.00	999.00
0.47	851.20	921.20	958.30	979.90	988.30	993.80	996.80	998.40	999.20
0.48	859.40	926.90	962.00	980.30	989.80	994.70	997.30	998.60	999.30
0.49	867.40	932.40	965.60	982.50	991.10	995.50	997.80	998.90	999.50
0.50	875.00	937.50	968.80	984.40	992.20	996.10	998.10	999.10	999.60
0.51	882.40	942.40	971.80	986.20	993.30	996.80	998.50	999.30	999.70
0.52	889.50	947.00	974.60	987.90	994.20	997.30	998.80	999.50	999.80
0.53	896.20	951.30	977.20	989.30	995.00	997.70	999.00	999.60	999.90

TABLE 4—Continued

(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)
Loss in F	Loss on $1,000 when C, the competing ability, is damaged from 2 to 10 degrees more than F, the functional ability								
	C^2	C^3	C^4	C^5	C^6	C^7	C^8	C^9	C^{10}
0.54	902.70	955.30	979.50	990.60	995.70	998.10	999.20	999.70	999.90
0.55	908.90	959.10	981.60	991.80	996.40	998.40	999.30	999.70	999.90
0.56	914.90	962.60	983.60	992.80	996.90	998.70	999.50	999.80	1000.00
0.57	920.50	965.90	985.40	993.80	997.40	998.90	999.60	999.90	
0.58	926.00	969.00	987.00	994.60	997.80	999.10	999.70	999.90	
0.59	931.10	971.80	988.50	995.30	998.10	999.30	999.80	1000.00	
0.60	936.00	974.40	989.80	996.00	998.40	999.40	999.80		
0.61	940.70	976.90	991.00	996.50	998.70	999.50	999.90		
0.62	945.20	979.20	992.10	997.00	998.90	999.60	999.90		
0.63	949.40	981.30	993.10	997.50	999.10	999.70	999.90		
0.64	953.40	983.30	994.00	997.90	999.30	999.80	1000.00		
0.65	957.20	985.10	994.80	998.20	999.40	999.90			
0.66	960.70	986.70	995.50	998.50	999.50	999.90			
0.67	964.10	988.20	996.20	998.80	999.70	1000.00			
0.68	967.30	989.60	996.70	999.00	999.70				
0.69	970.30	990.80	997.20	999.20	999.80				
0.70	973.00	991.90	997.60	999.30	999.80				
0.71	975.70	993.00	998.00	999.50	999.90				
0.72	978.10	993.90	998.30	999.60	1000.00				
0.73	980.40	994.80	998.60	999.70					
0.74	982.50	995.50	998.90	999.80					
0.75	984.40	996.10	999.10	999.80					
0.76	986.20	996.70	999.30	999.90					
0.77	987.90	997.30	999.50	1000.00					
0.78	989.40	997.70	999.50						
0.79	990.80	998.10	999.70						
0.80	992.00	998.40	999.70						

TABLE 5.

Standard of Measurement for determining Damages to F, the functional ability of the body. (Unit a, factors h, i k,.)

Loss in F, the functional ability of the body, in anchylosis of				Loss in F, the functional ability of the body, in amputation of			
Fingers,		Toes,		Fingers,		Toes,	
Little,	0.01	Little,	0.005	Little,	0.01	Little,	0.005
Ring,	0.02	Fourth,	0.005	Ring,	0.02	Fourth,	0.005
Middle,	0.02	Middle,	0.005	Middle,	0.03	Middle,	0.01
Index,	0.02	Second,	0.005	Index,	0.04	Second,	0.01
Thumb,	0.03	Big,	0.01	Thumb,	0.05	Big,	0.02
Wrist,	0.10	Ankle,	0.10	Hand,	0.20	Foot,	0.20
Elbow,	0.15	Knee,	0.15	Forearm,	0.25	Leg,	0.25
Shoulder,	0.15	Hip,	0.15	Arm,	0.30	Thigh,	0 30

TABLE 6—STANDARD OF MEASUREMENT FOR LOSS OF SIGHT.

Standard of measurement for determining damages to F, the functional ability of the body, for the loss of sight in one eye. (Unit g, factor w.)

(1) Degrees of Disability	(2) Scientific Standard	(3) Economic Standard	(4) Loss to	(5) Loss to F.
Slight loss of sight in one eye.....	1° from 0.7 to 0.6	from 6/6 to 5/6	1/6	from 0. to 0.03
	2° from 0.6 to 0.5	from 5/6 to 4/6	2/6	from 0.03 to 0.06
Severe loss of sight in one eye.....	1° from 0.5 to 0.4	from 4/6 to 3/6	3/6	from 0.06 to 0.09
	2° from 0.4 to 0.3	from 3/6 to 2/6	4/6	from 0.09 to 0.12
Nearly total loss of sight in one eye	1° from 0.3 to 0.2	from 2/6 to 1/6	5/6	from 0.12 to 0.15
	2° from 0.2 to 0.1	from 1/6 to 0	6/6	from 0.15 to 0.18

Total loss of sight in one eye.....0.18

FOR BOTH EYES.

1° slight loss of sight in one eye with 1° slight loss of sight in the other eye.....from 0. to 0.06
1° slight loss of sight in one eye with 2° slight loss of sight in the other eye.....from 0.03 to 0.09
2° slight loss of sight in one eye with 2° slight loss of sight in the other eye.....from 0.06 to 0.12
1° slight loss of sight in one eye with 1° severe loss of sight in the other eye.....from 0.06 to 0.12
1° slight loss of sight in one eye with 2° severe loss of sight in the other eye.....from 0.09 to 0.15
1° slight loss of sight in one eye with 1° nearly total loss of sight in the other eye.....from 0.12 to 0.18
1° slight loss of sight in one eye with 2° nearly total loss of sight in the other eye.....from 0.15 to 0.21
1° slight loss of sight in one eye with total loss of sight in the other eye.....from 0.18 to 0.21
1° severe loss of sight in one eye with 1° severe loss of sight in the other eye.....from 0.12 to 0.18
1° severe loss of sight in one eye with 2° severe loss of sight in the other eye.....from 0.15 to 0.21
1° severe loss of sight in one eye with 1° nearly total loss of sight in the other eye.....from 0.18 to 0.24
1° severe loss of sight in one eye with 2° nearly total loss of sight in the other eye.....from 0.21 to 0.27
1° severe loss of sight in one eye with total loss of sight in the other eye.....from 0.24 to 0.27
2° severe loss of sight in one eye with 2° severe loss of sight in the other eye.....from 0.18 to 0.24
2° severe loss of sight in one eye with 1° nearly total loss of sight in the other eye.....from 0.21 to 0.27
2° severe loss of sight in one eye with 2° nearly total loss of sight in the other eye.....from 0.24 to 0.30
2° severe loss of sight in one eye with total loss of sight in the other eye.....from 0.27 to 0.30
1° nearly total loss of sight in one eye with 1° nearly total loss of sight in the other eye from 0.24 to 0.30
1° nearly total loss of sight in one eye with 2° nearly total loss of sight in the other eye from 0.27 to 0.33
1° nearly total loss of sight in one eye with total loss of sight in the other eye.....from 0.30 to 0.33
2° nearly total loss of sight in one eye with 2° nearly total loss of sight in the other eye from 0.30 to 0.36
2° nearly total loss of sight in one eye with total loss of sight in the other eye.....from 0.33 to 0.36

Total loss of sight in both eyes..... 0.36

Note.—This table 6, Standard of Measurement for Damages to F, the functional ability of the body, for the loss of sight, is confined exclusively to the scientific and economic standard of measurement of central acuity of sight, which is the first and most important indispensable element of vision.

The second indispensable element of sight is the field of vision which is provided for in Table 6 (a), Standard of Measurement for Damages to F, the functional ability of the body, for the loss of any part of the binocular field of vision. The field of vision embraces half a circle or 180°, divided into six zones of 30° each. Slight loss of the binocular field of vision embraces its concentric contraction of 30°, or its equivalent in irregular contraction from 180° to 120°. Severe loss of the binocular field of vision embraces its concentric contraction of 60°, or its equivalent in irregular contraction from 180° to 60°. Nearly total to total loss of the binocular field of vision embraces its concentric contraction of 85°, from 180° to 10°, when it becomes total loss of functions for economical purposes. The damage to F, the functional ability of the body, is the same for the field of vision, as for the acuity of vision as given in Table 6 for these designations, that is, for slight loss of the field of vision it would extend to 0.12; for severe loss of the field of vision it would extend to 0.24; and for nearly total to total loss of the field of vision it would extend to 0.36.

The third indispensable element of sight is the muscular movements of the eyes which are classified in Table 6 (b), Standard of Measurement for Damages to F, the functional ability of the body, for the loss of the functions of any one or all of the extrinsic and intrinsic muscles of two normal eyes. In complete irremediable paralysis of the external rectus of one eye, causing double vision so the eye had to be excluded from taking part in binocular vision, the damage to F, the functional ability of the body, would be the same as the loss of the sight of that eye, namely, 0.18. In complete paralysis of all the extrinsic and intrinsic muscles of both eyes, the damage to F, the functional ability of the body, would be 0.36.

Provisions are made for all the losses in the field of vision, and in the muscular movements, which are readily comparable with the losses in the central acuity of vision.

TABLE 7—STANDARD OF MEASUREMENT FOR THE LOSS OF HEARING.

Standard of measurement for determining damages to F, the functional ability of the body, for the loss of hearing in one ear. (Unit g, factor w).

(1) Degrees of Disability	(2) Scientific Standard	(3) Economic Standard	(4) Loss to	(5) Loss to F.
Slight loss of hearing in one ear.....	1° from 0.7 to 0.6	from 6/6 to 5/6	1/6	from 0. to 0.02
	2° from 0.6 to 0.5	from 5/6 to 4/6	2/6	from 0.02 to 0.04
Severe loss of hearing in one ear.....	1° from 0.5 to 0.4	from 4/6 to 3/6	3/6	from 0.04 to 0.06
	2° from 0.4 to 0.3	from 3/6 to 2/6	4/6	from 0.06 to 0.08
Nearly total loss of hearing in one ear.....	1° from 0.3 to 0.2	from 2/6 to 1/6	5/6	from 0.08 to 0.10
	2° from 0.2 to 0.1	from 1/6 to 0	6/6	from 0.10 to 0.12

Total loss of hearing in one ear..... 0.12

FOR BOTH EARS.

1° slight loss of hearing in one ear with 1° slight loss of hearing in the other ear.....from 0. to 0.04
1° slight loss of hearing in one ear with 2° slight loss of hearing in the other ear.....from 0.02 to 0.06
2° slight loss of hearing in one ear with 2° slight loss of hearing in the other ear.....from 0.04 to 0.08
1° slight loss of hearing in one ear with 1° severe loss of hearing in the other ear.....from 0.04 to 0.08
1° slight loss of hearing in one ear with 2° severe loss of hearing in the other ear.....from 0.06 to 0.10
1° slight loss of hearing in one ear with 1° nearly total loss of hearing in the other ear.....from 0.08 to 0.12
1° slight loss of hearing in one ear with 2° nearly total loss of hearing in the other ear.....from 0.10 to 0.14
1° slight loss of hearing in one ear with total loss of hearing in the other ear.....from 0.12 to 0.14
1° severe loss of hearing in one ear with 1° severe loss of hearing in the other ear.....from 0.08 to 0.12
1° severe loss of hearing in one ear with 2° severe loss of hearing in the other ear.....from 0.10 to 0.14
1° severe loss of hearing in one ear with 1° nearly total loss of hearing in the other ear.....from 0.12 to 0.16
1° severe loss of hearing in one ear with 2° nearly total loss of hearing in the other ear.....from 0.14 to 0.18
1° severe loss of hearing in one ear with total loss of hearing in the other ear.....from 0.16 to 0.18
2° severe loss of hearing in one ear with 2° severe loss of hearing in the other ear.....from 0.12 to 0.16
2° severe loss of hearing in one ear with 1° nearly total loss of hearing in the other ear.....from 0.14 to 0.18
2° severe loss of hearing in one ear with 2° nearly total loss of hearing in the other ear.....from 0.16 to 0.20
2° severe loss of hearing in one ear with total loss of hearing in the other ear.....from 0.18 to 0.20
1° nearly total loss of hearing in one ear with 1° nearly total loss of hearing in the other ear...from 0.16 to 0.20
1° nearly total loss of hearing in one ear with 2° nearly total loss of hearing in the other ear...from 0.18 to 0.22
1° nearly total loss of hearing in one ear with total loss of hearing in the other ear.....from 0.20 to 0.22
2° nearly total loss of hearing in one ear with 2° nearly total loss of hearing in the other ear...from 0.20 to 0.24
2° nearly total loss of hearing in one ear with total loss of hearing in the other ear.....from 0.22 to 0.24

Total loss of hearing in both ears..... 0.24

TABLE 8—STANDARD OF MEASUREMENT FOR THE LOSS OF SMELL.

Standard of measurement for determining damages to F, the functional ability of the body, for the loss of smell. (Unit g, factor w).

(1) Degrees of Disability		(2) Scientific Standard	(3) Economic Standard	(4) Loss to	(5) Loss to F.
Slight loss of smell	1°	from 0.7 to 0.6	from 6/6 to 5/6	1/6	from 0. to 0.02
	2°	from 0.6 to 0.5	from 5/6 to 4/6	2/6	from 0.02 to 0.04
Severe loss of smell	1°	from 0.5 to 0.4	from 4/6 to 3/6	3/6	from 0.04 to 0.06
	2°	from 0.4 to 0.3	from 3/6 to 2/6	4/6	from 0.06 to 0.08
Nearly total loss of smell	1°	from 0.3 to 0.2	from 2/6 to 1/6	5/6	from 0.08 to 0.10
	2°	from 0.2 to 0.1	from 1/6 to 0	6/6	from 0.10 to 0.12
Total loss of smell.....					0.12

FOR THE LOSS OF TASTE.

(1) Degrees of Disability		(2) Scientific Standard	(3) Economic Standard	(4) Loss to	(5) Loss to F.
Slight loss of taste	1°	from 0.7 to 0.6	from 6/6 to 5/6	1/6	from 0. to 0.01
	2°	from 0.6 to 0.5	from 5/6 to 4/6	2/6	from 0.01 to 0.02
Severe loss of taste	1°	from 0.5 to 0.4	from 4/6 to 3/6	3/6	from 0.02 to 0.02
	2°	from 0.4 to 0.3	from 3/6 to 2/6	4/6	from 0.03 to 0.04
Nearly total loss of taste	1°	from 0.3 to 0.2	from 2/6 to 1/6	5/6	from 0.04 to 0.05
	2°	from 0.2 to 0.1	from 1/6 to 0	6/6	from 0.05 to 0.06
Total loss of taste.....					0.06

FOR THE LOSS OF FEELING.

(1) Degrees of Disability		(2) Scientific Standard	(3) Economic Standard	(4) Loss to	(5) Loss to F.
Slight loss of feeling	1°	from 0.7 to 0.6	from 6/6 to 5/6	1/6	Depends upon the extent of the area involved and the functions of the part or parts which have lost the sense of feeling.
	2°	from 0.6 to 0.5	from 5/6 to 4/6	2/6	
Severe loss of feeling	1°	from 0.5 to 0.4	from 4/6 to 3/6	3/6	
	2°	from 0.4 to 0.3	from 3/6 to 2/6	4/6	
Nearly total loss of feeling	1°	from 0.3 to 0.2	from 2/6 to 1/6	5/6	
	2°	from 0.2 to 0.1	from 1/6 to 0	6/6	
Total loss of feeling.....					do.

NO. 9—EVOM TABLE

Based on the premises that F, the functional ability, multiplied by T, the technical ability, multiplied by C, the competing ability, equals to E, the earning ability of a person, and that the gross economic value of man is the present value of all his earnings for a prospective working life, and that the net economic value of a man is the present value of all his earnings less the present value of all his personal expenses for his prospective life.

The money values here given may be used either for the gross, or the net, economic value of man, depending upon whether $1 per day is the gross, or the net, income per day for three hundred days of the year, namely $300 per year, for a prospective working life.

This table is computed on a 3½ per cent. discount basis.

At the Age of	Money Value	Annual Increase	Percentage of Increase in 5 years	No. living at	No. Deaths in 5 yrs.	Percentage of death in 5 years
Birth	133.29			513		
5	1388.44	251.03	911.31	372	141	27.48
10	2900.98	302.50	108.94	355	17	4.57
15	4754.26	370.65	63.88	346	9	2.53
20	5797.72	208.69	24.05	335	11	3.17
25	6114.51	63.35	5.46	321	14	4.17
	Decrease	Decrease	Decrease			
30	5985.71	25.76	2.10	307	14	4.36
35	5664.43	64.25	5.36	291	16	5.21
40	5262.30	80.43	7.13	275	16	5.49
45	4784.29	95.62	9.08	257	18	6.54
50	4178.96	121.04	10.57	237	20	7.78
55	3420.67	151.68	18.14	215	22	9.28
60	2413.00	201.52	29.45	189	26	12.09
65	1141.72	254.25	52.68	156	33	17.46
70	19.10	224.73	98.33	118	38	24.35
75	—609.32	125.68	3293.50	79	39	33.05
80	—1017.88	81.71	67.05	44	35	44.30

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ETIOLOGY AND TREATMENT CHRONIC CATARRHAL OTITIS MEDIA.

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In a former paper read before the American Laryngological, Rhinological and Otological Society, published in 1909, I stated that, "there are more causes for deafness, (1) than the interference with the passage of sound to the terminal filaments of the auditory nerves, and (2) the interference with the transmission of sound from the terminal filament to the brain. We may have fatigue—a state of weakness, suspension of or inability to transmit its special stimulus, or to interpret that stimulus correctly. As, in the eye we may have an asthenopia or weakness of vision, so we may have a similar condition in the ear. We may have functional disturbances due either to internal or middle ear disease. Occasionally we meet with a case, the symptoms of which would indicate middle ear diseases; in another group, the symptoms indicate disease of the internal ear, yet a careful examination reveals that neither disease exists; hence, we must look elsewhere for the cause. I have in several instances found the existence of Bezold's Symptom—complex of oto-sclerosis, namely (a) increased bone conduction, (b) negative Rinne, (c) elevation for lower tone limits, and yet this disease did not exist. Since in the eye we may have diminished or obliteration of functions from toxemia, why we may not for the same reason have a similar condition in the ear? That we do meet with toxemia of the auditory apparatus I have frequently demonstrated. In this class of cases, I find some disorder of the nervous, digestive, excretory and circulatory systems which has intensified a comparatively trivial irregularity of the ear into troublesome deafness."

From the observations made prior to and since the above statements were published, I deduce from the records of

one hundred cases that the etiology has been one of three of the following:

First: Repeated attacks of exudative catarrh of the middle ear and Eustachian tube, the result of nase-pharyngeal catarrh or nasal-pharyngeal adenoids.

Second: Over-indulgence in the use of saccharin foods, meats, coffee, tea, alcohol and tobacco.

Third: Rheumatism and anemia.

In a few instances the cause of the disease could be traced to other conditions, but these three were by far the more frequently met with.

The question of climate in which the patients live was a small factor in the etiology as compared with how he lived and what was the chief article of food and drink. Climate has much to do towards producing a predisposition to both of these, but further than this, I do not believe it a potent factor to be considered.

When the conditions which interfere with the mechanism of the Eustachian tube and the ventilation of the middle ear have been removed, and all purulent diseases of the nasal accessory sinuses, carious teeth, septic inflammation of the supra-tonsillar fossa have been relieved, and the disease still continues, then the relief is to be obtained through hygienic and systemic treatment. This I believe otologists have not regarded as important and necessary in as large a proportion of cases as my own observations would lead me to conclude: We should *not know less of otology*, but as Otologists, we *should know* more of physiology, especially that of the ear and its relation to the body-complex. Ankylosis and adhesions of the ossicular chain of the middle ear cavity should be treated as we treat similar conditions in other portions of the body.

In the latter class of cases we know that local treatment is a secondary factor toward the relief and the primary cause must first be dealt with. Since the cause is from within the body, and not due to external conditions, the cure or alleviation must come largely through removing these causes

through the employment of dietetic and hygienic measures and the correction of such habits as predispose to or aggravate the existing conditions.

Especially is this true where the disease continues after the removal of bands of adhesions caused by atrophied adenoid tissue which had resulted in interference with the ventilation of the middle ear cavity through the Eustachian tubes.

Hence systemic treatment first; local measures, second: I have found the causes of these conditions to be the one which results in faulty metabolism, as evidenced by imperfect and insufficient elimination, the result being toxema or deposits in the ossicular joints resulting in sclerosis or an oedema and thickening of the lining of the middle ear cavity.

The most prominent *subjective* symptoms met with were impaired hearing for speech, although the hearing for high tones and sharp shrill noises was unimpaired, this degree of deafness being more intensified during mastication.

The hearing power is generally better in the morning than in the evening, and in dry weather than on damp rainy days. Tinnitus, almost invariably, although frequently intermittent and aggravated by mental excitement, paracusis Willisi frequently complained of. Pain in the ear is seldom noticed but there is a feeling of fullness often accompanied by mental depression. All these symptoms are aggravated by any systemic or mental condition which lowers the body vitally, such as worry or fatigue.

There is frequently an excessive loss of bone condition compared with the high degree of hearing power for speech, though in many instances a marked loss of aerial conduction was found, when tested by a wide range of tuning forks. Frequently musical tones are less adversely affected than noises.

The *objective* conditions consist of thickening, opacity or retraction of the membrana tympani with evidence of fixation of the ossicular chain and the partial stenosis of the Eustachian tube. In many cases the entire membrane was

thickened and of a dense white color and some had calcareous deposits due to lime salts on the tympanic mucosa.

In a large number of cases the membrana tympani was relaxed, presenting a flabby appearance. Also in a number of cases the Eustachian tubes were apparently more than normally patulous.

In deciding upon a line of treatment for such cases we have described laboratory tests are to be made use of to assist in arriving at the etiology. A careful urinalysis is made for indican, excess of uric acid, urea and phosphates; the blood pressure taken, and the blood count made to ascertain the amount of hemaglobin. Careful investigation of the patient's diet is important and if there be found any particular article of food used more freely than others, completely exclude it for a few days to ascertain what effect it may be having in causing the faulty metabolism. The etiology of the disease is often difficult and unsatisfactory and a diagnosis is arrived at only by exclusion of other causes.

Two well known aphorosims are here most applicable: First, "a chain is no stronger than its weakest link." Second, one part of the body cannot be treated to the exclusion of the whole,—for one part cannot be involved in a grave pathological or functional disturbance without the entire body suffering in consequence. Admitting the truth of these statements in the disease of the middle ear, the weakest link in the chain, is this organ, and any excessive task placed upon the remainder of the body will first be manifested here, and the existing condition intensified and aggravated.

While it is true that the primary cause in the majority of my cases could be traced to the sequellae of naso-pharyngeal disease, yet frequently after these were relieved entirely, the disease not only duly persisted but became intensified, deafness and tinnitus increasing. In equally as large a number of cases I could find no evidence of these sequellae, but on the contrary a well marked lithemic or neurotic condition existed, both of these being due to a

faulty metabolism the result of intestinal autotoxemia. Most of these cases lead a sedentary life which in turn leads to a sluggish elimination. The excessive use of sweets is a greater task upon the lymphatic system than any other article of food, and the eating liberally of meat produces more uric acid than any other. In dealing with this condition, much harm is done by overtreatment of the ear. The membrana tympani is made more relaxed by too frequent use of the Politzer bag. Hyperemia of the middle ear cavity and Eustachian tube is increased by too frequent and too irritating applications to these regions. Mobility of the ossicular chain by massage and instrumental manipulation, I believe, does more harm than good. The chief factors guiding me in the treatment of this disease are:

First: The removal and prohibition of the etiologic factors.

Second: Elimination of all accumulated irritants.

Third: The rehabilitation and preservation of function.

* * * * *

First: The removal and prohibition of the etiologic factors.

(a) Under this head I would prohibit all indulgence in red meats, pastries and concentrated saccharine foods; the use of alcohol, coffee and tea,—except in small quantities. One important part I wish to emphasize is that oftimes it is the combination of food that does harm rather than any particular article of food itself, therefore a most restricted diet is prescribed consisting mainly of vegetables, fruit and butter-milk; while meat of fowl and eggs I have not found objectionable.

Where butter-milk cannot be obtained, the lacticbacillary tablets have proved an effective substitute.

(b) The removal of unsanitary surroundings and the encouragement of hygenic living.

(c) The establishment of daily physical exercise.

Second: Elimination of all accumulated irritants.

(a) The emptying and cleaning of the alimentary canal, a process which is by no means as easily accomplished as is generally supposed. My usual routine is to give 5 or 10 grains of calomel, followed in 6 or 8 hrs. by one ounce of castor oil,—this to be followed in three or four hours by ½ ounce Magnesium sulphate and one drachm Bicarb. soda in 6 ounces of carbonated water. After this, Magnesia sulph. one dr. is given daily before breakfast for from 2 to 4 weeks, the object being not only to empty the alimentary canal but to hyper-alkalienize the secretions.

(b) Bathing—the frequency and temperature of the daily bath being adjusted according to the individual temperament and need.

Third: The rehabilitation and preservation of function.

(a) With the articles of food and drink properly adjusted and hygienic living established,—there still remains for a time a certain amount of medication necessary to the rehabilitation of functions weakened by more or less constant irritation from the various causes referred to—and over varying periods of time. First among such measures in my practice has been the use of some form of alkaline-alterative such as iodide of soda or iodide of strontium 1 to 5 grains; salicylate of sodium or strontium 5 to 10 grains, and 10 drops of tinct. nux vomica well diluted, t. i. d. immediately after meals. If there be a deficiency in hemoglobin some ferrugious preparation is given.

(b) Local: (1) Removal of naso-pharyngeal and faucial abnormalities which are in themselves obstructing or thro their purulent or necrotic retentions leading to toxic absorption.

(2) Keeping the naso-pharyngeal membrane clean thro the use of a non-irritating alkaline wash—administered by means of a nasal cup two or three times a day.

(3) Stimulation and equalization of the circulation—both blood and lymphatic—around and within the tympanic cavity, thro the use of vibratory massage with moderate pressure over the subglenoid fossa; gentle inflation of middle

ear cavity with a stimulating vapor of chloroform, iodine or menthol two or three times a week.

Only in cases of marked retraction and actually adherent membrana tympani to employ the use of suction manipulation with the Siegal otoscope.

Where the membrane is very relaxed and flabby and paracusis Willisi is plainly noticeable, I have in some cases noted improvement by painting the posterior inferior quadrant of the membrane with tincture of iodine (25%) once or twice a week; in others by painting with flexible collodion in the same location.

This should be used cautiously and discontinued as soon as more than temporary discomfort is observed.

The results of the treatment have been satisfactory only in proportion to the degree of faithfulness with which these measures were adhered to.

Relapses often recurred when the patient returned to his former method of living. The results however, have often been so gratifying to the patients that no further admonition was necessary especially after they have been warned by one or more relapses and return of deafness, tinnitus and excessive secretion.

In several cases I have been surprised to find improvement in these conditions following the administration of small doses of thyroid extract aid stimulation of internal secretions. I am unable to explain its physiological or therapeutic action, but good results were very apparent.

Good results have also been seen after administration of thiosonaime for some weeks, in cases of well defined adhesions.

CONCLUSIONS.

Of the 100 cases treated in ten years, 50 of which I have been able to keep in sufficiently close touch to know the results of treatment, in 40% of these there was an increase of 10% to 50% in bearing; in 25% there was relief of all subjective symptoms, and while the hearing had not decreased, there was no appreciable improvement.

In the remaining 35% there was improvement only during treatment.

Discussion.

Dr. Thomas J. Gallaher of Denver, Col., referred to an article by Mr. Charles J. Heath of London, in which the author suggested a new treatment for chronic catarrhal otitis media accompanied with paracusis Willisi. Mr. Heath has achieved some remarkable results in this type of deafness. He gives a most explicit and convincing explanation of the cause of paracusis.

Dr. E. E. Holt, Portland, Maine, said he was very much interested in Dr. Stucky's paper and felt that he could agree with him in practically every essential. He did not wish to say anything to detract from its excellence, but simply to discuss an incident referred to as a quality of the hearing ability of some of the patients tested, namely, that condition known as paracusis Willisiana. Dr. Holt referred to a paper on Boiler-Maker's Deafness and Hearing in a Noise, which he read before this Society just thirty years ago at its meeting at Lake George, in which he said:

"From the tests and observations of all who claimed they could hear better in a noise, it was evident that conversation as carried on in the shop could be heard better by them than by those who had better or normal ears; but when in testing these cases the voice was used in the same tone as in a quiet place or any of the other tests were employed, they were never heard better in the noise. The results of my tests of those who have made the claim that they could hear better in a noise, has shown me that they were inexact in their observations and labored under a misapprehension of facts, which they were generally willing to admit when shown their error, for without any exception I have never found a person out of more than a hundred examinations that a noise improved the hearing power in the least degree. We find not infrequently in the literature of Otology, reference made to the subject as though it was an established fact that a noise in

some unknown way improved the hearing power in certain individuals, but, so far as I am aware, it is not stated that the writer or any one else ever made a careful investigation to ascertain whether the claim of such persons was a real one or not.

"There has been no lack of explanations why a person heard better in a noise, but none of them have been generally accepted. The most important point, however, whether it was an actual fact that the noise did improve the hearing power in certain pathological conditions of the ear, as it appears to do, has generally been left out of the question. Von Tröltsch is the only author who raises this question. He says: 'Deaf persons very often tell you of hearing better in the midst of noise and roaring sounds. Misapprehension and lack of proper observation are generally at the basis of these statements.' As all my tests have been negative in regard to persons hearing better in a noise, it occurred to me that the apparent phenomenon could be explained by comparing the normal ear with the pathological conditiion, which is assumed to exist in the ears of persons who make this claim. The external and middle ear collect and conduct the sound waves inward to the internal ear, where they are received and thence transmitted by the nerve to the brain and there interpreted as sound. In examining this remarkable process somewhat in detail, we find that the sound waves are collected, condensed and reflected inward by the auricle through the meatus to the membrana tympani, setting it and the ossicles which are connected with it and one with the other by delicate joints, into a to-and-fro motion, which movement is communicated through the foot-plate of the stirrup bone to the fluid of the labyrinth where it is received by the terminal filaments of the auditory nerve and thence transmitted inward by that nerve to the sensorium to be interpreted as sound. It is evident then, if this be the normal physiological process of hearing, that when the ear is trying to receive the impressions of particular waves of sound, others causing, or attempting to cause, an additional movement of these ossicles must necessarily interfere with

the transmission and reception of the waves of sound listened for, and somewhat confuse the listener, as in fact they do. From the history of repeated attacks of inflammation of the middle ear and naso-pharynx, the pathological condition of the ear under consideration may be assumed to be rigidity of the articulations of the ossicles and secondary tympanic membrane covering the fenestra rotunda, and consequently increased resistance to the conduction of sound through the tympanic cavity. This would be a condition of the ear in which more or less of the waves of sound given off in any place would not be received—constituting the deafness—and if more or less of the waves of sound made no impression upon the conducting apparatus of the ear there certainly would not be so much disturbance in such an ear in a noisy place like a boiler-maker's shop, a mill in operation or a railway car in motion, as there would be in the normal ear. The noise producing less disturbance in such an ear and the voice being invariably raised in a noisy place the waves of sound produced by it are conducted by the sound transmitting apparatus of the ear to the labyrinth with less confusion than in the normal ear, in which the ossicles must be in a constant to-and-fro movement or else the innumerable noises would not be heard and there would be no confusion of sound. Hence it may follow that persons whose ears are in the pathological condition here assumed to be present, may hear conversation as carried on in a noise better than the normal ear, and better than they hear ordinary conversation in a quiet place. But to repeat, when the same tone of voice was adopted in a noisy place that was used in a quiet one, or the tick of the stop-watch, vibrations of the tuning fork and snap of the finger nails were employed for ascertaining the hearing power they were never heard better in a noisy place, and in most cases not as well as in a quiet one; in other words, the noise never improved the hearing power in any case that has come under my observation."

Drs. O. D. Pomeroy, H. D. Noyes, R. C. Brandeis, John Green and Samuel Theobald who were present and discussed

this paper accepted the explanation given, but Dr. D. B. St. Roosa, who was not present, took sharp exceptions to the conclusions reached in Dr. Holt's paper after it was published, and promised to show Dr. Holt some cases in which the hearing was actually improved for a given sound in the midst of a noise. Dr. Roosa, however, was never able to show a single case to Dr. Holt, and finally admitted that the improvement of hearing of certain persons in a noise was not due to any increase in the hearing but to the fact that they heard the voice, when necessarily raised in a noise, better than they heard it as used in carrying on a conversation with them in a quiet place, and above all, that they heard better in a noisy place than those with normal ears. In all the thirty years since reading this paper Dr. Holt had never found a person, claiming that he could hear better in a noise, who by tests actually heard a given sound better in a noise than in a quiet place. He, therefore, would like to establish the fact that the true definition of paracusis Willisiana is a condition of the ears of a person whose hearing for conversation as carried on in a noisy place is best, and better than those with normal ears under these conditions, but that noise does not aid any person to hear a given sound better than he hears that sound in a quiet place.

Dr. Holt added that the explanation he had given was the reason why in the case referred to by Dr. Woods the hearing was best in the noise with the voice raised sufficient to carry on conversation under such conditions.

Dr. Sohier Bryant, of New York City, suggested that a better result would be obtained with contractile collodion than with the flexible variety. He has tried the application of cantharidin through the canal, as suggested by Heath, of London. The results did not appear to be any more certain than irritation of the tympanum by vibratory massage or intra tubal injections of irritating substances.

THE RELIEF OF TINNITUS BY THE USE OF NITRATE OF SILVER APPLIED WITHIN THE EUSTACHIAN TUBE.

By WILLIAM C. BRAISLIN, M. D., Brooklyn, N. Y.

The systematic application of nitrate of silver within the Eustachian tube has proved in my experience with the method of application here described to be of great usefulness, particularly in tinnitus due to swelling and engorgement of the mucous membrane lining the tube. This is recommended as an addition to the treatment by the catheter and other methods.

Incidentally, I may refer to some other means which I have employed to relieve congestion by the tube. I have occasionally used during the last four years a small extensible knife, which I had fitted into the lumen of a catheter-shaped canula, by means of which blood letting from the mucous membrane of the mouth of the tube has been performed with beneficial results in some cases. Likewise, I have employed the method of breaking up adhesions about the mouth of the tube with the finger, as recommended by Dr. Logan at the meeting of this society in 1909. Injections of solutions into the tube with the flexible stiletted bougie have also been used. During the past winter I have furthermore systematically employed the applications of silver in cases of tubal catarrh, O. M. C. C. and mixed middle and internal ear cases; in short, all classes of non-suppurative ear cases in which swelling of the tube was an evident accompaniment of the other conditions present. For the most part these applications have been made by small cotton pledget, twisted on a flexible wire applicator, dipped in the silver solution and carried through the lumen of the catheter into the Eustachian tube for the distance of a quarter of an inch to one inch. This is done at the finish of the ordinary catheter inflation and before removing the catheter from its position. For greater convenience, previous to the beginning of inflation through the

catheter, the wire is wrapped and laid within easy reach beside the bottle of solution. On finishing the inflation, the wire is dipped into the solution and carried through the lumen of the catheter into the Eustachian tube, the patient meanwhile swallowing, if necessary, to facilitate its easy passage. The application made, the wire is at once withdrawn. A 5% solution of nitrate of silver is usually employed, which, when acute inflammation is present in the tube, creates a sharp, but only temporary, sensation of pain.

Instead of appending here a considerable series of cases of the various ear diseases referred to above, treated by the method of combined inflation and nitrate of silver applications within the tube, I decided that a few summarizing statements would better economize time, and serve the purpose of this communication equally well. I have proved to my own satisfaction that the relief of the tinnitus is more rapid and effectual where silver applications within the tube are employed as an addition to catheterization. Several recurrent or chronic cases so treated have been under observation and occasional treatment for from three to ten years. In some of these cases the patient generally obtained greater relief in the same time than formerly. Tubal swelling is nearly always accompanied by tinnitus, and it is a relief to the patient to get improvement from tinnitus of tubal origin even should other symptoms be unrelieved.

No result is expected by this method in the treatment of tinnitus due to immobility of the stapes, from tinnitus due to labyrinthine or auditory nerve lesion or to that of the nervous centres or of vascular origin. Tinnitus due to digestive, circulatory or general diseases is not appropriately treated alone by local method, but congestion in, and about, the tube is in part relieved by local applications.

In nearly all of these cases, information as to the actual conditions about the mouth of the tube has been derived from the pharyngoscope, chiefly, however, in detecting some causes of tinnitus which might possibly have been overlooked, such as posterior hypertrophies of the turbinates, small polypi impending against the tube, specific ulcers and others.

Tinnitus which is due to inflammatory conditions of the tube, seems to the writer, to yield more rapidly and permanently when nitrate of silver applications within the tube are added to the other methods of treatment. The presence of diseases of the tube must be regarded as an indication or tendency on the part of the sufferer to acquire more serious ear lesions. All contributory influences in the nose and pharynx are to be eradicated as stringently as in ear diseases of a more advanced character; otherwise, recurrence will be proportionally earlier and more severe.

The applications of silver within the tube have the advantage over other and more elaborate methods of treating the tube in the slight additional time required to apply the treatment so that it can be employed systematically, if necessary, daily. The writer believes its usefulness to be superior to the use of the plain or unmedicated bougie. Its systematic application in his own experience has proved more generally useful than the use of the metal or gold bougie for carrying the electric current.

Discussion.

Dr. B. Alexander Randall, of Philadelphia, said he had long held the view, now generally accepted, that the most frequent cause of tinnitus is stuffiness of the Eustachian tube. Upon relief of this partial stenosis of the tube depended relief of the tinnitus. To that end he had employed dionin, as used in ophthalmological practice. Those who had tried the method would not abandon it. The bougie, for this purpose, he had almost laid aside. He simply injected, with a catheter, a two to five per cent. watery solution of dionin, using sufficient force to cause it to penetrate the tympanic cavity. One or two repetitions succeeded, with rare exceptions, in effecting freeing of the tube with relief of noise and usual gain in hearing. He had been able to bring about a marked gain in cases which had been given up by others as being beyond hope. Dionin had proved of more value in his hands than any other one measure.

In reply to questions from members of the Society, Dr. Randall said dionin is ethyl morphia hydrochloride, which had been used as a substitute for heroin and other agents which relieve cough. He had seen no general physiological effect from it. It should, however, be used with extreme caution, especially in the stronger solutions. There was very little pain after its injection. One patient had said she lost consciousness, but she was able to go home unattended.

The action of dionin was largely that of stimulation of circulation and lymphatic absorption. It was a wonderful absorbent.

Dr. Hiram Woods, of Baltimore, corroborated what Dr. Randall had said concerning dionin. He had used it at the latter's suggestion, in a number of cases which showed loss of low tones, and (imaginary or) real improvement of hearing in noisy places. He had used it by injection through a catheter. He cited the case of a woman, with very stubborn tinnitus, in the right ear. In one ear her low limit was 512 V. S., in the other 256 V. S. She had a motor boat, and had observed that when she sat near the engine she could hear conversations which, at home, she could not hear two feet away. In the course of two months' treatment with injections of dionin twice a week, she could hear conversations with much greater ease. The tinnitus had markedly disappeared. He had never seen any pain follow the injection. He did not know how to explain its action except that it produces hyperemia somewhat similar to so-called Bier's hyperemia. Used for two or three months it would relieve chronic symptoms in middle ear conditions.

Dr. S. MacCuen Smith, of Philadelphia, confirmed in his experience the statements of Dr. Randall concerning dionin. He had met with some failures, but they were in cases in which this agent was not suitable. Cases in which there was congestion of a circulatory type would be relieved. He would be rather fearful of using nitrate of silver in such strength in the tube, especially since he was accustomed to using dionin without reaction.

Dr. Braislin, in closing the discussion, said he had used nitrate of silver hundreds of times in the tube in the strength given, and even stronger, without producing any reaction at all. Its chief effect was astringent. He was led to use nitrate of silver in making the ordinary application of silver in nasopharyngeal work. The rapidity and ease with which solutions could be applied with the cotton tipped wire gave it an advantage over other methods. Tinnitus was improved by getting the astringent inside the tube. In using the pharyngoscope, the presence of adhesions binding the cartilage of the tube to the wall of the pharynx may be observed. The application of astringents to the Eustachian tube has seemed an agency of decided results in lessening tinnitus and in improving the hearing.

He wished to express his sense of obligation to Dr. Randall in so generously advancing, in connection with this paper, his experience in the use of dionin, in cases similar to those in which the writer of the paper has used astringents with similarly beneficial results.

THE VALUE OF SERUM INJECTIONS IN THE TREATMENT OF HEMORRHAGE.

By FRANCIS R. PACKARD, M. D, Philadelphia, Pa.

I wish to present a report of two cases of hemorrhage occurring in patients who were undoubtedly true hemophiliacs, which was checked in each instance, after other means had failed, by the subcutaneous injection of serum.

W. M., Hospital, No. 1225, a white boy, aged 11 years, was admitted to the wards of the Pennsylvania Hospital, July 10, 1911. When he was brought to the Hospital his mother made the statement, of which I was, however, not informed until after the operation, that a doctor had warned her that the child had a tendency to bleed and should never be operated upon except in case of absolute necessity. The mother informed me personally, also after the operation, that on three occasions she had been obliged to take the boy to a hospital in order that bleeding from splinter wounds or slight cuts might be stopped. There was no history of any other hemophiliac in the family.

The boy's physical examination previous to operation was negative.

On July 11, 1911, the day subsequent to his admission, he was given ethyl chloride, followed by ether, and I dissected out his tonsils, which were large and cryptic, with a knife and scissors. I removed a large soft adenoid with Lowenburg's forceps. There was very little bleeding throughout and immediately after the operation. The boy reacted well. During the night following the operation, he began to have considerable oozing from the right tonsillar region and from the nasopharynx.

The next day, July 12th, a large hematoma appeared in the pharynx. Pressure, the application of cold to the neck, and adrenalin solution to the bleeding areas, failed to affect the bleeding in any way.

The next day, July 13th, there were extensive submucous hemorrhages all over the palatal surface. Twenty cc. of plain horse serum, made by a well known firm, was injected subcutaneously on this day. The next day there was very little change in his condition, and the horse serum was once more injected in the same quantity. The next day, the 15th of July, the breath became very foul, evidently from decomposed submucous clots in the pharynx and nasopharynx. He vomited a considerable quantity of blood on various occasions, and his bowel movements contained quite large quantities of dark blood. The injections of horse serum were continued and he was given calcium lactate, 3 grs., every four hours. On the 17th of the month, two days later, he was in very bad condition. His pulse was rapid and thready, skin leaky, and there were numerous new hemorrhages throughout the mucous membrane of his mouth. On this day Dr. Longcope, the pathologist of the Pennsylvania Hospital, with whom I had conversed about the case, suggested the use of freshly prepared rabbit serum, made in the Hospital, as he thought possibly the commercial horse serum might not be efficient. Accordingly an injection of 20 cc. of rabbit serum was given, a very short time after it had been drawn in the laboratory of the Hospital, and from that date the patient had no further hemorrhages whatever. His condition steadily improved under stimulation with strychnia and the administration of iron and large quantities of easily digested food, and he was discharged from the Hospital cured, on the 7th of August, 1911.

The second case in which I have been a witness to the efficiency of the injection of serum for the checking of hemorrhage, was that of a boy ten years old, one of whose uncles and his grandfather had died of hemorrhage following a slight injury, and one of his first cousins died from hemorrhage after the removal of tonsils and adenoids.

The father had consulted me as to the removal of this boy's tonsils and adenoids, which were very large, but I declined to operate upon him in view of the history narrated above.

Two years later, in June, 1910, the boy's family physician, Dr. Branson, of Rosemont, Pa., called me up on the telephone and told me that the boy had been bleeding continuously for a week following the removal, by a dentist, of one of his molar teeth. Various local and constitutional measures, such as packing the cavity with bone wax, and the use of calcium chloride had been tried without any avail. I suggested the use of plain serum by injection. Some horse serum was obtained and 20 cc. injected into the buttocks, whereupon the hemorrhage promptly ceased and never recurred.

I have reported these two cases because it seems to me that they are both of them undoubtedly true hemophiliacs in whom hemorrhage was stopped by the injection of serum. Theisen 1. (Footnote 1. Annals of Otology, Rhinology and Laryngology, September, 1911,) and Hubbard 2. (Footnote 2. Annals of Otology, Rhinology and Laryngology, March, 1910.) have both considered the subject in recent articles of timely value. The use of serum injections was urged by Weil, who suggested using only 1 cc. of the serum at the first injection in order to avoid anaphylactic danger. In the author's cases much larger doses were used with no ill results. The serum should be as fresh as possible and administered with every aseptic precaution. It has been suggested that diphtheria antitoxin might be used in emergencies where it was impossible to procure plain serum. In view of the number of deaths which have been reported, occurring in patients suffering with respiratory disorders, but not having diphtheria, from the prophylactic use of diphtheria antitoxin, my personal belief is that the use of diphtheria antitoxin would be attended by a certain amount of risk. I believe, however, in view of the danger of immediate death from hemorrhage, that the risk would be justifiable if the plain serum were unobtainable.

Discussion.

Dr. E. Terry Smith, of Hartford, Conn., has had considerable experience with the rabbit serum. At first he em-

ployed antitoxin, but found it of very little value, but in a large number of cases he has found fresh rabbit serum of great value in controlling hemorrhage, especially in hemophiliacs. Dr. C. T. Beach usually prepares the serum, and the method he employs is as follows:

The animal is anesthetized with ether and the whole body moistened with a one per cent. lysol solution to keep the hairs from flying. An incision is made from the angle of the jaw to the sternum and the jugular vein exposed. Two hemostats are then placed close together as far down in the neck as possible and the vein cut between them. The upper part of the vein is then dissected out and a conducting tube for the blood, about an inch long, is so formed. The distal end of this is then held over the collecting tube and freely incised, permitting the blood to escape. When a tube is full the flow is stopped, either by rotating the hemostat or exerting steady traction upon it. The bleeding is continued until the animal is exsanguinated. A three and a half pound rabbit will yield forty to fifty c. c. of blood from which nearly thirty c. c. of serum can be obtained. The collecting tubes are allowed to stand until the clot forms, then a platinum needle is passed around the clot to free it from the walls of the tube, then the tube is centrifugalized and the clear serum is drawn off with a bulb pipette and transferred directly to the syringe and is then ready for injection, no preservative being added.

Dr. Frederick L. Jack, of Boston, cited a case of hemorrhage, from so slight an operation as opening the drum—in which serum injection was successfully employed. An ordinary paracentesis had been performed, followed, two hours later, by considerable hemorrhage from the ear. He was called, and applied bandage and gauze on the outside. In the night he was again called and found the patient covered with blood down to the waist and the pillow soaked. The hemorrhage continued for several days, despite treatment. The mastoid became involved, and operation seemed imperative. Rabbit serum was suggested, and two vials, each con-

taining 20 c. c. of rabbit serum were procured. The contents of one vial was injected in the afternoon, in the back. During the night the patient did so well, that the second injection was omitted. Operation was uneventful. Several days afterwards, however, hemorrhage began again. Another injection of serum was given, and all bleeding ceased. Rabbit serum was apparently effective in this case, and Dr. Jack would use it again in similar conditions, rather than the horse serum.

Dr. James F. McKernon, of New York city, has had some experience with thrombo-kanouse. In seventy-five per cent. of cases the powder acted very well when applied over bleeding surfaces.

Dr. William K. Rogers, of Columbus, Ohio, cited a case in which hemorrhage of three days' duration followed the removal of adenoids and tonsils. He suggested to the surgeon the use of the rabbit serum. The serum was employed without success. Careful exploration of the pharynx revealed a large tag of tissue which had been left, and which was evidently the mechanical cause of the hemorrhage. Such possibilities should be borne in mind.

AUTHORS' INDEX

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ERNEST DeWOLF WALES.

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